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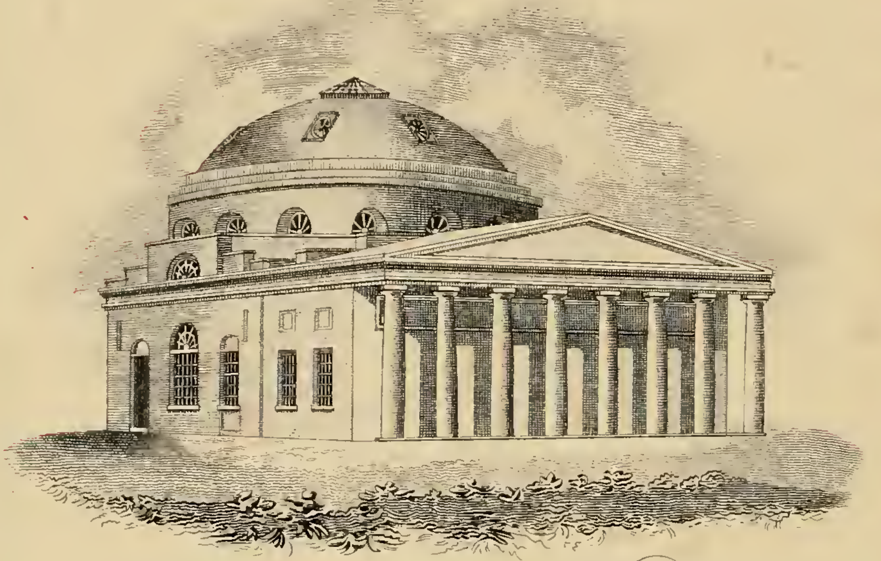

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APPENDICITIS: SOME OBSERVATIONS BASED ON A REPORT OF 1,245 OPERATED CASES*†

ARTHUR M. SHIPLEY, M.D.

Appendicitis is a disease that manifests itself in many different ways. The story of the condition is a varied one; the symptoms may be misleading, the position of the appendix is not constant, and the disease may be very acute or very chronic with all possible gradations between the two extremes. Some portion of the appendix may be in intimate relationship with almost any structure in the abdomen while, on the other hand, referred symptoms from many other tissues may point to the region of the appendix. The symptoms and signs may be masked by fat or by a phlegmatic or cheerful patient, and the disease behaves differently in children, in young and middle-aged adults, and in the old.

The bacteriology of the infection is complicated and confusing. The incidence of the disease is certainly not receding and, in spite of everything that has been said and written, the mortality is slowly on the increase and much higher in the United States than in most European countries. It is a disease of civilized peoples. Physicians dealing with primitive races rarely see appendicitis.

In acute appendicitis most of the efforts of the medical profession are directed at early diagnosis and early operation, the danger of catharsis and the advantages of no food and rest in bed. These facts have been impressed upon medical students and physicians, but in most instances of perforated appendicitis the fault should be laid at the door of the patient himself, who does not call a physician until the damage is done.

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* From the Department of Surgery, School of Medicine, University of Maryland.

† Read at the Annual Meeting of the Medical and Chirurgical Faculty of Maryland, Baltimore, April 26, 1939.

In two large American cities an organized campaign has been carried on with success in the schools, stressing the folly of disregarding abdominal pain and the taking of cathartics. Attempts at impressing the adult population have been more or less futile. The fight against the rising death rate in appendicitis will probably be a part of the Youth Movement.

There has been a very large recent literature on the subject of appendicitis and this has been confusing to both physicians and laymen. This controversy on the part of surgeons deals with only one phase of the subject and has to do with the classification and treatment of peritonitis complicating acute appendicitis with perforation. The problem of the medical profession, the community and the individual is to get the patient to the surgeon before perforation takes place.

The mortality figures are quite uniform in the many reports and are very convincing. In chronic, recurrent and early acute appendicitis before peritonitis develops, the death rate is less than one-half of 1 per cent, while in peritonitis complicating appendicitis the death rate, especially in late diffuse peritonitis, may be as high as 40 per cent.

In a recent paper by Ransom from Coller's Clinic at the University of Michigan, ninety-two papers on the subject of surgical intervention in appendiceal abscess and spreading peritonitis caused by appendicitis are reviewed and there is a striking unanimity as to mortality figures, as can be seen from the following percentages collected from the different reports: 21.1 per cent, 22.8 per cent, 24.7 per cent, 27.55 per cent, and 21.5 per cent in diffuse peritonitis; in localized abscess the death rate is 4 per cent, 5 per cent, 7.3 per cent, and 4.2 per cent. These figures tell the whole story of the difference in opinion and practice as to time of operation in late peritonitis complicating perforated appendicitis.

In 1902, Ochsner in this country, and about the same time (1905) Sherren in England, advocated the waiting treatment in late diffuse peritonitis in the hope and expectation that the spreading peritonitis would recede and localize.

Statistics on the subject are not entirely accurate, however, unless proved by operation or autopsy. A recent case in the University Hospital is illuminating. A young man in a nearly moribund condition, with the diagnosis of perforated appendicitis and late diffuse peritonitis, was admitted. He was dehydrated, there was acid-base imbalance from vomiting, his abdomen was hard, distended and silent, and his general appearance was one of extreme toxemia. He was treated and improved somewhat, but died without being operated on and at autopsy there was nothing wrong with the appendix. The peritonitis was the result of the presence of a pneumococcus without any lesion in the chest.

In considering the sources of infection in diffuse peritonitis that might be mistaken for perforation of the appendix, the following possibilities do not add to the patient's outlook or the surgeon's peace of mind: slowly leaking gastric or duodenal ulcer, the less fulminating cases of acute pancreatitis, perforation of the gallbladder, Meckel's diverticulitis, rupture of a salpinx or perforation of the uterus, peridiverticulitis with perforation, and perforation of malignant or tuberculous ulceration of the intestines. The author has seen three cases of perforated diverticulitis of the colon and three cases of perforated malignant ulceration of the gut within the last twelve months.

It is a pity that the surgeon ever has to consider the above-mentioned problems in considering the treatment of appendicitis. He should see the patient before perforation takes place. When an individual has persistent pain in his abdomen, he should see his physician; and when a physician sees a patient with abdominal pain and he believes that he is dealing with appendicitis, he should send such a patient to a surgeon. "It is a mistake to be influenced by the very considerable mass of literature that might lead him to wait," Osler said long ago, "there is no medical treatment for appendicitis." It is perfectly true that many cases of acute appendicitis will subside, if treated by rest, with nothing given by mouth, no catharsis, and an ice cap on the abdomen, but the mortality is confined almost entirely to the cases that do not subside.

"It is also true that a localized mass following an attack of acute appendicitis may disappear without operation, but again quite a number of them go on to suppuration and disaster. If all cases of appendicitis were early ones when they came to the surgeon the problems and risks of peritonitis and drainage would largely disappear."

Barnes sent out a questionnaire recently to one thousand hospitals and received reports of 228,598 appendectomies performed in the twenty-five months between June 1, 1935 and July 1, 1937. Of 208,885 nonperforated appendectomies there was a mortality of 0.8 per cent. Of 13,965 perforated appendices the mortality was 18.3 per cent.

The real problem in dealing with a patient with symptoms indicating appendicitis is to determine, first, whether the suspected appendicitis is acute, chronic or recurrent. If chronic or recurrent, there is ample time to carefully consider all phases of differential diagnosis from other chronic conditions in the abdomen, in the lower chest or in the spine.

In acute cases the differential study should be made promptly.

Mistakes may occur by not considering carefully the chest, the postperitoneal structures and the pelvis. The author's own experience warrants him in believing that a sufficient amount of time spent in arriving at a fairly accurate diagnosis of acute appendicitis is amply justified by feeling reasonably secure in attacking the diseased appendix through the McBurney approach.

If the acutely inflamed appendix is full of pus or is the site of widespread thrombosis in its wall or is gangrenous but not yet perforated, there will usually be considerable peritoneal reaction, such as fresh adhesions, solid exudate covering the appendix and seropurulent liquid exudate, either localized or spreading, in the peritoneal cavity. The surgeon should attempt a careful, painstaking removal of the appendix with as little handling as possible and with no contamination of the edges of the abdominal wound. Raw surfaces should be peritonealized and all fluid sucked from the peritoneum. If the appendix is not perforated when the abdomen is opened and is not torn by handling, it is safe to close the peritoneum without drainage. At times it is wise to drain the abdominal wall or to close it very loosely. There is no more important decision that the surgeon is called on to make than how and when to drain. Spreading peritonitis hastened by lack of drainage and mechanical obstruction resulting from the presence of drains are both death-dealing complications.

If peritonitis is believed to be present before operation, it is well to try to separate these cases into two groups, early and late. The English and others speak of acute appendicitis, which is not subsiding, as one day, two day, three day, four day, etc. and in this way attempt to arrive at some decision as to immediate operation or the Ochsner-Sherren treatment. The whole picture is confused because it is difficult, at times, to determine just when the appendicitis attack began and when the appendix perforated. A deeply lying or postperitoneal appendix makes it most difficult to evaluate the time factor. Probably a better method of determining whether the patient has an early or late peritonitis before operation is by a careful consideration of symptoms, the duration of nausea and vomiting, if either is present, the amount of rigidity and distention, the degree of paralytic ileus, retention of urine, and the water and acid-base balance, together with pulse and temperature reading, as well as the appearance of the patient as determined by the degree of toxemia.

If the peritonitis is an early one, immediate operation offers the best outlook even though peritoneal drainage may be necessary. If the peritonitis is late, the outlook is bad in any case. Death may be attributable to a number of causes, but these causes are all set in motion by the peritonitis. The causes of death may be divided into three groups. In one, the fatal outcome is the direct result of the peritonitis itself, dehydration, exhaustion, toxemia, paralytic ileus, infected abdominal wound, pyelphlebitis, etc. In another, death is caused by some complication outside the abdomen, such as pneumonia, empyema, infarction and septicemia, and in the third group mechanical intestinal obstruction usually caused by the angulation of loops of small intestine in the neighborhood of the drainage tract creates a serious menace to life. This form of obstruction has a mortality in excess of 50 per cent.

Differentiation between paralytic ileus and mechanical obstruction is sometimes very difficult. To operate for paralytic ileus is still further to harm the small intestine already dangerously distended, and to fail to operate promptly on mechanical obstruction is to invite disaster. The patient, his relatives, house officers and surgeon go through an uneasy period until the issue is settled.

The statements made in this paper are based on a study of 1245 patients with appendicitis treated in the author's service in the University Hospital since June 1, 1930. Appendectomies done while operating for some other condition in the abdomen are not included.

There were 558 operations for chronic or recurrent appendicitis, with two deaths; 367 patients with acute appendicitis, but without noticeable peritoneal reaction, with two deaths; 163 patients with suppurative, thrombotic or gangrenous appendicitis without gross perforation, but with early peritonitis, who were not drained, with one death; 42 patients with early peritonitis without gross perforation whose peritoneum was closed, but the abdominal wall drained, with no deaths; 4 cases of necrotic appendicitis with early peritonitis whose peritoneum was drained because of uncertainty as to a safe closure of the stump, with no deaths; 49 cases of late localized peritonitis, with two deaths, and 62 patients with late diffuse peritonitis caused by perforation, all of whom were drained, with twenty-one deaths.

Altogether, there were 925 patients with no noticeable peritoneal reaction with four deaths, a mortality of .043 per cent, and 320 patients with varying degrees of peritoneal infection with twenty-four deaths, a mortality of 7.5 per cent. In order to stress the importance of the time factor in peritonitis this figure of 7.5 per cent should be compared with the devastating mortality in late diffuse peritonitis—62 cases with twenty-one deaths, 34 per cent mortality. The relatively low mortality in the group with late localized peritonitis—49 patients with two deaths—should also be noted as both of these deaths resulted from intestinal obstructions.

The striking contrast in these mortality figures between late diffuse and late localized peritonitis is the basis of the Ochsner-Sherren treatment. The expectant method is not so successful, however, in children and the old, because in the extremes of life resistance to infection is relatively poor and the waiting period for localization to take place is likely to be hazardous. In young adults and middle-aged individuals, however, and especially in the fat-filled abdomens of many persons past forty, the whole picture of spreading peritonitis will often change for the better with waiting and appropriate treatment directed at vomiting, paralytic ileus, dehydration, sympathetic nerve disturbance, pain and apprehension.

An analysis of the twenty-eight deaths is instructive. There were four deaths in the 925 clean cases. One was an anesthetic death in a patient with a chronic rheumatic heart, who was thought to be a good general anesthetic risk as his heart was well compensated. Of the other three deaths, one was caused by pulmonary embolism and two by pneumonia. Of the twenty-four deaths in the peritonitis group, seven were attributable to mechanical intestinal obstruction. The one death in the 163 patients with early peritoneal involvement resulted from pneumonia.

In the winter and spring, many patients come in with symptoms of rapidly developing appendicitis and with some respiratory tract infection as well. They are a source of great anxiety, because of the symptoms of early pneumonitis and the risk of inspiration anesthesia. Most of these patients, unless they are children, are operated on under spinal anesthesia.

Consideration of the above classification and the causes and percentage of deaths in the various groups leads naturally to a number of conclusions, all of which have been stressed repeatedly in the literature. A patient with appendicitis may have some other illness that modifies the risk and influences the choice of anesthetic. In many instances patients with bronchitis, influenza, emphysema, pulmonary tuberculosis, persons with poor liver or kidney function, and individuals with some cardiac disability will do better if operated on under spinal anesthesia. The choice of the anesthetic and care in its administration may be more important than the method of operating.

Most of the deaths result from late diffuse peritonitis and there is a very extensive literature dealing with this subject that should be studied by any surgeon operating in this field. To say that these deaths would be eliminated if early diagnosis and early operation were the uniform rule is to repeat a very trite surgical axiom, but in spite of repetition and admonition patients are still admitted to hospitals with late peritonitis caused by a perforated appendix.

The occasional death from embolism is a reminder of the wisdom of confining the operation to the region of the appendix, together with gentleness and painstaking care in the details of the operation itself. There is rarely any necessity for roughness and great speed in operation for appendicitis.

The two most common late complications of abdominal surgery are incisional hernia and postoperative adhesions. The McBurney incision or some modification of it will almost entirely remove both of these risks, provided drainage is not done; if drainage is necessary, the material for drainage can be placed to good advantage through the McBurney incision in most of the regions that it is desired to drain, especially into the lateral gutter of the ascending colon, into the pelvis or down to the region of the head of the cecum.

There are two conditions that are almost impossible of differential diagnosis from acute appendicitis before operation, Meckel's diverticulitis and acute diverticulitis in the cecum. The important thing is not to overlook either, especially if the symptoms and signs are more marked than the appearance of the appendix seems to warrant.

"Perforation is accepted by most surgeons as on the borderline between mild and severe peritonitis, but the issue is determined by the kind, number and virulence of the bacteria and the resistance of the host. Recently considerable attention has been paid to the kind of micro-organisms present. Study of the bacteriological reports reveals a wide divergence in the kind and number of organisms recovered from the unruptured, acutely inflamed appendix and from the pus of peritonitis complicating appendicitis. There are fairly uniform reports as to the aerobes, Escherichia coli (Bacillus coli) and various strains of streptococci being found in a high percentage of cases. Other aerobes are found as well; the staphylococcus, a gram-positive bacillus, and others. Occasionally Hemophilus influenzae (B. influenzae) is present.

"There are a number of reports as to the presence of anaerobes. Altemeier and Bower et al. differ sharply as to the presence of Clostridium welchii (B. aerogenes capsulatus, B. perfringens, B. welchii), but other observers are in accord with the latter in reporting a high incidence of clostridia, especially Cl. welchii. Another clostridium occasionally present is Cl. oedematis maligni (Vibrion septique, Cl. septique, bacillus of malignant edema). Altemeier reports for the first time the presence of B. melanogenicum in 92 per cent of patients examined. Bower et al. also report that 69 per cent of patients suffering from or recovering from spreading peritonitis had demonstrable and significant amounts of circulating antitoxin to Cl. welchii. Jennings found Cl. welchii in the lumen of the appendix in 90 of his cases.

"Altogether, these bacteriological studies of pus in peritonitis, complicating the perforated appendix, are very disturbing to the surgeon. Most operators formerly believed that they were dealing with Esch. coli, different strains of streptococci and an occasional staphylococcus, and many suspected that H. influenzae played an unknown role in peritonitis and appendicitis.

"But the knowledge that anaerobes are commonly present adds another factor that demands consideration. Under certain conditions, parasites attacking the tissues of the body prepare a pabulum in which the saprophytes flourish, and in addition to this the question of symbiosis demands immediate consideration. This is pertinent when it is recalled that progressive gangrenous ulceration of the abdominal wall is found in association with drainage tracts following perforated appendicitis, especially when it is remembered that this is a symbiotic infection between specific strains of the staphylococcus and streptococcus.

"Meleney has stressed the possibility of symbiosis as an explanation of the varying behavior of suppurative peritonitis: 'In any specific prophylactic or active treatment for peritonitis one must take into account the symbiosis of the commonest organisms found in the peritoneal exudate, namely Esch. coli, the green streptococcus and Cl. welchii'." (Shipley, 9.)

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HAY FEVER*†

The Present Status of Treatment

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INTRODUCTION

The first mention of symptoms resulting from contact with plants dates back to 1565, when Botallus (1 a) called attention to symptoms displayed by patients exposed to roses and musk. Early in the nineteenth century, Bostock (1 b) of England described a condition that occurred seasonally and which, he stated, the public in general thought was caused by the effluvium of hay and with which opinion he disagreed. In two papers read before the Medical-Chirurgical Society of London, the first on March 16, 1819 (1 b) and the second on April 22, 1828 (1 c), this author clearly described for the first time the condition we know as hay fever.

Despite Bostock's delineation of this symptom complex as a definite clinical entity he and those following him, except for a few references to the possibility that pollens might play a part, considered the heat of summer as the causative agent of the condition until Blackley's work in 1873. Blackley lived in Manchester, England and was, himself, a sufferer from hay fever. After fourteen years of the most arduous research, in which he used himself as the test subject, this worker published one of the most complete reports in the history of medicine (1 d). Seven years later he published a revised and enlarged volume (1 e) that is a monument to his industry and established all of the essential features of the condition as we know it today. Amongst these was the use of the skin test as a method of diagnosis. In view of this latter fact, it is amusing to learn that a modern columnist, praising the virtues of an excellent American medical school a few years ago, stated that its scientists had discovered a method of diagnosing specific sensitivities, namely, by putting a little of the powdered substance upon the patient's skin; if he was sensitive, a reaction would develop attesting to the fact and revealing the offending agent.

Following Blackley's proof of the rôle of pollen in the causation of hay fever, his work was gradually disregarded and several theories,

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since relegated to oblivion in favor of his conception, had their brief day.

One interesting exception was the work of Dunbar (1 f). This observer confirmed the work of Blackley and, because of the furor then being created by the new antidiptheritic serum, injected animals with a pollen extract that he prepared. His idea was that this material was a true toxin and that it could produce an antiserum of therapeutic value. It was called pollantin and had quite a vogue for a while. An interesting sidelight in this regard and one that demonstrates the lability of medical knowledge is given in a recent publication. Thommen (1 g) in 1931, after discussing the claims for pollantin and the counter claims for another somewhat similar product, made this statement: "This episode is rather interesting when viewed in the light of our present knowledge concerning the utter lack of therapeutic value of both these sera."

In 1935, Cooke and his coworkers (2, 3) offered evidence to support a new theory to the effect that treated hay fever patients develop a specific factor that protects them from the pollen. Furthermore, they reported the successful treatment of hay fever patients by means of the passive transfer of these antibodies. This work has been recently confirmed and extended by Langner and Kern (4). Thus, even though the production of an immune serum in animals has not been accomplished, relatively the same result has been achieved in humans and renders the hypothesis of Dunbar far from unreasonable.

The modern treatment of hay fever began with Noon's report (1 h) upon his experience with the active treatment of the condition with pollen extract. After his death the work was carried on by many men and has been continued until now the literature upon the subject is quite voluminous.

With the advent of a scientific and effectual procedure for the amelioration of the condition, interest was aroused in the subject with the result that a considerable fund of knowledge has been gained, not only as regards its treatment but as regards other phases of the disease. Certain of the more important of these will be briefly discussed. A reasonably complete but by no means exhaustive bibliography is appended for the use of those interested in the subject who may desire to investigate further certain phases of the condition.

DIAGNOSIS

As in all allergic conditions, the diagnosis of hay fever involves two problems: first, the nature of the condition and whether it is hay fever, and secondly, the identification of the causative pollen or pollens.

Usually, if one has a reasonable knowledge of the local wind pollinated plants causing hay fever symptoms, the first of these questions presents little difficulty in definitely seasonal cases which are the ones considered in this article.

The determination of the offending pollens is commonly considered a relatively simple problem, depending as it does upon the characteristic allergic procedure of skin testing. However, certain observers call attention to the fact that there is a wide discrepancy between the clinical picture and the skin test findings in many cases. Efron (5) reported a large number of false positive and false negative tests, and suggested the introduction of dry pollen into the nares in doubtful cases. He is supported in this by Blumstein (6), who calls attention to the fact that the test was used by Blackley in his original work upon hay fever.

Harten (7) and his associates routinely tested 100 atopic patients who did not exhibit the symptoms of hay fever and found fifty-five clearly positive to pollen. They concluded that active sensitization of the skin occurs in a varying percentage of exposed atopics who possess the hereditary predisposition to pollen sensitization. This would seem to suggest also that the intranasal test is essential to proper diagnosis in many cases.

Tuft (8), on the other hand, states that positive skin tests practically always are important in hay fever although false negatives may occur and require mucous membrane tests, i.e., conjunctival or intranasal application of the suspected pollens. This probably represents the opinion of the majority of allergists who depend upon the skin tests but do the mucous membrane tests when they seem necessary for purposes of confirmation.

TREATMENT

Innumerable plans for the control of pollen hay fever have been suggested by different workers from time to time. The most important and most satisfactory is the conventional hyposensitization method commonly used. In view of work recently done, which will be discussed later, it would seem to be based upon a demonstrable immunological mechanism. Before presenting this major procedure, however, it would seem advisable to discuss briefly some other forms of therapy that have received considerable publicity at various times.

Endocrine substitution therapy. The relationship between endocrinopathy and hay fever is so little understood, even though there are some facts that seem to suggest that it plays possibly a fundamental part in the production of the allergies, that Tuft's (9) position seems a sound one, namely, to withhold endocrine substitution therapy unless there is definite evidence that there is a need for it. In other words, endocrine products should not be used unless the evidence of specific endocrine inadequacy is clear-cut, in which event substitution therapy is directed toward the correction of that condition.

Acid-base alteration therapy. Beckman (10) in 1929 suggested the use of nitrohydrochloric acid in the treatment of allergic conditions. In his opinion the production of a slight degree of acidosis is beneficial in these cases and he gives the results of 185 hay fever patients treated in this manner by twenty-six cooperating physicians. They obtained marked or complete relief in 66 per cent of the cases as contrasted with 70 per cent relief obtained by five allergists treating 2,185 cases in the conventional way. He cites as an advantage of his method that none of the patients in his group experienced unpleasant reactions, such as those occurring when the injection method with pollen extract was used.

In 1930 (11) Beckman made a further report in which he stated that he was much more impressed with the efficacy of the acid treatment. However, he modified his method by suggesting the use of dilute hydrochloride acid, ammonium chloride or sodium acid phosphate in place of the original strong acid; in addition, he advocated acid foods and the avoidance of alkaline foods, such as fruits and greens.

In accord with Beckman's views as to the need of acid in allergics are the findings of Criep and McElroy (12), who reported the finding of achlorhydria and hypoacidity in a large percentage of atopics and suggested the advisability of further investigation of the effect of acid therapy.

Loveless (13), in an attempt to evaluate conclusively this form of treatment, studied 138 patients most carefully. Her conclusions were, amongst other things, that there was no decrease in gastric acid in allergics; on the contrary, the "mean free acid was higher in the allergic group than in the normal". She also found that the duration of allergic symptoms was not a factor that influenced gastric acidity; no influence upon acid-base balance was demonstrable by chemical studies; and that no impressive clinical response resulted from acid therapy. Her cases were studied by proper statistical methods and her conclusion was that, "The number of allergic patients who obtained definite and sustained relief is probably insignificant if one considers the possibilities of psychic influence and the law of chance".

This work was so carefully done that it is difficult not to feel that the burden of proof is definitely upon the proponents of the method.

Calcium, phosphorus, potassium, sodium and vitamin D therapy. For years the idea has been prevalent that there is a decrease in the blood calcium in allergics and that its administration was of material aid in the control of these conditions. This idea was supported by Pottenger in 1924 (14). In 1928 Criep and McElroy (12) studied this theory by blood chemistry determinations and concluded that there was no lowering of calcium in allergics and that the giving of calcium did not permanently affect the blood concentration. Sterling, quoted by Crandell and Feinberg (15), claimed to find a lowering of the blood phosphorus in these cases two years later.

In 1930 Ramirez (16) studied fifty cases each of asthma, hay fever and urticaria, picking those that showed an initial blood calcium below 10 mg. per 100 cc. of blood. Even though he succeeded in increasing the blood finding his conclusion was that, "The administration of calcium, or calcium and phosphorous did not influence the symptoms or course of the disease in a single case". He did not find a uniformly decreased calcium in these cases.

Greenberg and Gunther (17) studied the amount of diffusible calcium present in allergics and its relationship to the total figure and found no deviation from the normal average.

Whether or not the blood phosphorous was lowered as reported by Sterling was the basis of studies made by Crandell and Feinberg (15). They found normal values and no clinical benefit from its use.

Because of the persistence of the impression that calcium was of benefit in allergic conditions, and despite the negative reports listed above, Rappaport (18) and his associates began the use of massive doses of vitamin D for the purpose of raising blood calcium concentrations. They reported favorable therapeutic results, especially when the method was accompanied by conventional injection methods.

At the present time it would seem that most observers are disposed to be skeptical regarding the benefit to be derived from this type treatment but feel that further study is indicated.

Recently Bloom (19) advanced the concept that allergy is, in part at least, a "disturbance of electrolyte metabolism". He suggests, therefore, the administration of potassium in these conditions. As a basis for this he quotes articles that seem to indicate that there is a close connection between potassium and the action of adrenalin and that potassium is decreased in certain dermatoses. In addition, he uses it for its diuretic effect to lessen the local edema present in allergic shock organs during an attack. His reports are most favorable in certain cases, especially those with hay fever. Further trial will determine the value of this suggestion.

Ionization. In 1935, Warwick (20) suggested intranasal ionization as the treatment of choice in hay fever. He used a solution containing zinc, stannous sulphate and cadmium sulphate and claimed unusual therapeutic virtues for this combination. He also used it with excellent results in a lesser number of cases of asthma and hyperesthetic rhinitis. He claimed that all except one were relieved for a year or more. Since this original article, several workers have sought to evaluate the method (21, 22, 23, 24, 25, 26) and the consensus of opinion at the present time is that his method is of doubtful value and, because it is not free of danger, should not be used.

Elimination and avoidance of pollen. As would seem natural in a condition caused by an agent occurring in the air, such as hay fever represents, it was early realized that the elimination of pollen from the patient's environment or the removal of the patient to a pollen-free section was an object much to be desired.

As a result, hay fever resorts in different sections of the country became quite popular with those patients economically able to avail themselves of this luxury. As the areas in question were patronized, improvements were made which usually included the elimination of wooded areas, thus allowing the different pollen producing plants to gain a foothold and, eventually, destroy the value of the area for this purpose. Because the selection of a proper place was usually a more or less hit-or-miss proposition many patients failed to obtain benefit.

To overcome this difficulty, Durham (27, 28), using data derived from pollen counts made over a period of years, has evolved an index which denotes the value of any given section as a hay fever resort. He has mapped the entire country and presents his results so that they may be available to anyone interested in the problem of finding a low pollen area. Metzger (29) also stresses the necessity for knowing something of pollen conditions before advising climatic change for the relief of pollen sensitivity symptoms and explains how futile such change may be if lacking intelligent motivation.

The other approach mentioned, namely, the elimination of pollen from the patient's environment, naturally involved the use of filtration for the purpose of removing pollen from the air breathed by the patient. Cohen (30, 31), devised an air filter for this purpose and reported that hay fever patients began to improve in a few minutes after entering a pollen-free room and became symptom-free in from six to eight hours. Pollen asthmatics, on the other hand, took as much as several days to clear themselves of symptoms, according to his first report. In his later report, he revised this conception and noted that hay fever patients took from ten to twenty-four hours, while pollen asthmatics might take up to seven days to become asymptomatic.

Some years later, Nelson (32) and his associates made a more elaborate study along these lines and found that the results obtained were far from being perfect and varied, as one would expect, with the degree of sensitivity, pollen concentration and the weather. Cooled air was not as beneficial as natural temperature air because, in their opinion, of the increase in relative humidity.

From these reports one may conclude that the filtration of the air in the room occupied by the patient may be distinctly helpful in controlling symptoms but it cannot supplant the usual injection therapy if the patient intends to leave the controlled environment to pursue his usual activities.

A recent modification of the filtration method both for patients' rooms and for wear as a mask has appeared. It is based upon the process of electrostatic precipitation (33) and requires a potential of several thousand volts. Because of this the large units used for the clearing of pollen from the air in rooms seem quite practical; the small potential of 1½ volts available in the mask, however, makes it seem very probable that any benefit derived from the device results from mechanical filtration and is not dependent upon the minute amount of current available.

The foregoing presents the major plans offered from time to time for the treatment of hay fever and is intended simply as: a brief resumé; an attempt to assist in their evaluation; and the listing of a reasonably complete bibliography for those interested in further study of the subject.

Specific hyposensitization. This, of course, refers to the conventional method of treatment which is dependent upon the administration of increasing doses, parenterally, of the extract of the pollen or pollens incriminated in a given case. Unfortunately, the term desensitization has become firmly associated with the process and is generally used to designate it. This was attributed originally to the apparent analogy between anaphylactic desensitization in animals and the seemingly similar mechanism in treating pollen sensitivity. It was soon apparent, however, that although the object was the same, the degree of achievement in humans was so much less quantitatively than that in animals, that there must be basic differences between the two.

How great this contrast is becomes apparent immediately upon further consideration. A sensitized guinea pig, weighing about 300 grams, can be sensitized and then rendered completely insensitive by a second sublethal dose, or doses of the antigen. However, a human being, weighing 150 pounds, may be made tolerant to a dose of some hundredths of a milligram of the pollen involved but it is impossible to desensitize him to the point that he is able to tolerate even these amounts if the injection is made intravenously. The most that can ever be attained is to raise his tolerance to a point that will make him able to withstand the amount of pollen usually found in his environment. Fortunately, this is adequate from a practical standpoint in the vast majority of cases. Nevertheless, the results accomplished can hardly be designated by the same term as that used in animal anaphylaxis if false impressions are to be avoided; consequently, hyposensitization is the better term because it more nearly describes the results achieved.

No attempt will be made here to give the details of dosage and the conventional method of administration. This has been covered so thoroughly in the journals and in texts upon the subject, as Tuft's (34), and so much more adequately than would be possible in the limited space of an article of this type, that no useful purpose would be served. Only a brief outline of the steps leading up to the present status will be given.

Originally, preseasonal injections were thought to be the method of choice and, in fact, the only safe one available. Vaughn in 1922 treated one patient coseasonally with great caution and obtained excellent results. Ten years later, after considerably more experience with the method, he reported upon it most favorably (35). The consensus of opinion at the present time would seem to be that the administration of pollen extract should be continued through but not limited to the actual pollen season.

In 1927, Brown (36) suggested the year-round or perennial treatment and made a further favorable report in 1932 (37). Since then there have been many favorable reports (38, 39). Most workers in this field at the present time agree that the perennial administration of pollen extract is the most effectual plan and is the method of choice.

Variations of the foregoing that have not received general acceptance are: maximum pollen dosage as advocated by Brown (40); subcuticular administration, Duke (41); intravenous therapy, Lichtenstein (42); intradermal pollen therapy, Phillips (43); and oral route of administration, Black (44). The use of bee venom (Forapin) is suggested by von der Trappen (45) and good results claimed.

Hyposensitization. The parenteral administration of pollen extract remains the method of choice in the treatment of pollen sensitivities. Perennial or year-round injections, seemingly, are definitely more efficacious than when the treatments are given according to other schedules.

In the past, acceptance of the correctness of the foregoing statements was of necessity based simply upon the clinical results obtained. The persistence of positive skin tests without any decrease in the degree of sensitivity (46) thereof, and, in fact, with an increase in some patients even though their results were excellent, placed the proponents of the method upon the defensive.

In line with the present trend which is more and more placing allergy upon an immunological basis and, thus, subject to objective study, Cooke and his coworkers (2, 3) in 1935 demonstrated what would seem to be the underlying mechanism of hyposensitization. This work has been amply confirmed by Kern and his associates (4) just recently.

Because this represents one of the major advances in the field of allergy, it would seem important to describe it in detail. Before doing this a brief description of the allergic antibody and of the technic used in its demonstration will be given.

In the blood of skin sensitive patients there occur, invariably, antibodies designated by Coca as reagins. Their presence can be demonstrated by passive transfer to the skin of normal individuals and in no other way. Transference to animals, as is done with anaphylactic antibodies, is impossible. The technic of such transfer is relatively simple. Citrated whole blood or the serum from coagulated blood is introduced into the skin of a person previously proved to be skin test negative to the allergen or allergens to which the donor (patient) manifests sensitivity. After an incubation period of an hour or more (the usual is twenty-four hours) about 0.05 cc. of the extract to which the patient reacted positively is introduced into the exact site of the forementioned blood or serum injection of patient's blood into the normal recipient's skin. If the transfer is successful, there will result a positive reaction exactly similar to the reaction occurring in the skin test positive patient.

Because the allergen or extract has the power to temporarily exhaust the transferred reagins, a repetition of the test immediately upon the subsidence of the positive reaction results in failure. If, however, several days later the test is repeated at the site of the passive transfer, a positive reaction will result owing to the fact that the test material or allergen is a foreign body and is destroyed by the recipient. The transferred reagins, however, having attached themselves to the normal recipient's cells, will remain for several weeks.

Cooke (2, 3) discovered that if the blood of treated hay fever patients was used for the transfer, the results were quite different. His method involved a variation of that described above for passive transfer and consisted of mixing the reagin containing blood of the patient secured before hay fever treatment and that obtained after a thorough course, together with a measured amount of allergen or pollen extract. With the former mixture containing untreated patient's blood he obtained an immediate reaction, as would be expected. With the latter mixture containing treated patient's blood he obtained no reaction, which indicated that there was something in the blood that blocked the reaction.

A further variation from the usual was revealed by subsequent testing after the lapse of forty-eight hours. The pretreatment blood site failed to respond, as would be expected, but the post-treatment blood site responded and showed that the blocking substance had disappeared. He proved that this blocking substance was specific for the material under consideration, in this instance ragweed pollen extract, which had been used in treating the patient and to which the patient was sensitive.

In addition to the foregoing, Cooke (2, 3) demonstrated the presence of this blocking substance clinically. This was done by the transferrence of the immune substance from adequately treated hay fever patients to untreated patients, both by transfusion and by subcutaneous injections of serum. Hay fever patients who had received no other therapy and who had begun to have symptoms obtained a high degree of relief from the foregoing procedures.

Evaluation of hyposensitization therapy. Tuft (47) states that failure should be expected in from 5 to 15 per cent of all cases treated by this method. Of these he feels that faulty treatment is responsible in 10 per cent because of the following avoidable causes: faulty selection of extracts; faulty dosage control; failure to continue treatment coseasonally; use of impotent extracts; failure to recognize concomitant sensitizations to other allergens. He calls attention to a very important feature of the proper treatment, namely, the prevention of asthma caused by the pollen involved (48). Hyposensitization is very efficacious in this regard and decreases the incidence of this complication from a percentage variously estimated at from 30 to 50 per cent in untreated cases to a negligible percentage in those properly managed.

In an attempt to discover the percentage of cases that might be expected to obtain permanent relief, Vander Veer (49) studied his large group and arrived at the following conclusions: Permanent relief can be expected in well over 50 per cent of cases treated by specific pollen injections; those treated a number of years are more likely to experience such relief, especially those treated for from five to eight seasons; the final result will usually be better in those experiencing the best results seasonally; and the higher the pollen content of the air, the less likely is the result to be permanent.

SUMMARY

a. A brief resumé of the pertinent facts in the history of hay fever has been outlined.

b. Several forms of therapy, other than the conventional method of hyposensitization, have been discussed and the opinion of the majority of allergists as to their value has been given.

c. A reasonably complete bibliography has been presented for those desiring to pursue the general subject further or to go into any particular phase in greater detail.

d. The apparent demonstration of an immunologic basis for the relief achieved by hyposensitization has been presented and discussed.

CONCLUSION

1. The treatment of hay fever by the hyposensitization method is effectual and is the result of a demonstrable immunological mechanism.

2. The value of other methods of therapy, which have been suggested from time to time, has not been determined and their further study would seem to be necessary before final judgement can be passed.

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A REVIEW OF PROGNOSTIC CRITERIA IN MAMMARY CARCINOMA*†

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In order to review the clinical and histopathological prognostic factors in carcinoma of the female breast, 150 consecutive radical mastectomies were assembled from the files of the University Hospital and the rather formidable task of follow-up was undertaken. In these cases 94.6 per cent or 142 of them have been traced to January 1, 1939 or to death. Since the series of operations ended on November 4, 1933, all cases traced had at least a five-year follow-up period. Although numbering 150 cases, they occurred in 149 individuals. (See Case A.) They constituted all the original radical mastectomies for carcinoma of the female breast performed at the University Hospital from 1923 to 1933 inclusive.

Case A: A. D., a 62 year old, white woman had a radical right mastectomy by the late Dr. Frank Lynn in May, 1930. No regional metastasis was found histologically. The tumor was a scirrhous carcinoma located in the upper inner quadrant. She remained well until April, 1933, when a mass appeared in the upper inner quadrant of the left breast. A radical left mastectomy was done, with no metastasis found histologically. Although the positions of the tumors suggested that the second was a metastatic growth, it showed histologically a distinct papillary character entirely lacking in the first tumor. The patient has since remained quite well. It is believed that these were multiple primary tumors.

Of the 150 operations, eighty-four were performed by one surgeon, thirty-four by another, fourteen by still another, eleven by a fourth, two by a fifth and one each by five other surgeons. In all instances a more or less modified Halstead procedure was followed, with generous sacrifice of skin, a meticulous axillary dissection and occasional Thiersch grafting. The policy in general has been one of liberal interpretation of operability, as indicated by the facts that twenty-one of the cases were ulcerated and seventy-eight of them had palpable axillary or cervical nodes which were thought preoperatively to be metastatic.

OPERATIVE RESULTS

There were ten postoperative deaths in the group, viz., deaths occurring before the patients left the hopital. This constitutes an opera-

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tive mortality of 6.6 per cent. Because this seemed high an analysis was made of the causes of these deaths, with the following results. With one exception, all of the patients who died were beyond fifty years of age. Five of the ten had advanced ulcerated lesions. Two died of massive pulmonary emboli. Two others died of intercurrent disease contracted after operation and before discharge, one with a Type 4 lobar pneumonia, the other with a ruptured appendix. The other case died of uremia and was proved by autopsy to have had acute nephritis.

Of the 150 cases thirty-nine were living and apparently cancer-free after five years or more. There is no case in this series in which cancer is known to have reappeared after five years of freedom from symptoms. This represents 26 per cent actual five-year cures. Many series in the literature report results as five-year survivals. In this series there are ten additional cases living more than five years after operation, which would give a 32.7 percentage of five-year survivals. It should be noted here that the eight cases not traced are counted as failures (died of cancer before five years) in calculating percentages.

PRELIMINARY DIAGNOSTIC EXCISION OF TUMOR

There has been some protest made against excision of tumor and frozen section as a preliminary measure in doubtful cases of mammary neoplasm. Ample evidence is at hand to indicate that carcinoma can be easily transplanted during operative manipulation. A strikingly illustrative case which was recently encountered in this department was that of a woman with extensive peritoneal carcinosis whose ascites was relieved by paracentesis. Within a few weeks a nodule appeared in the abdominal wall at the site of puncture and after excision this was found to be a carcinomatous implant of the same morphology as the original tumor. In the meantime, malignant cells had been demonstrated to be unusually numerous in the centrifugate of the ascitic fluid.

In view of this quality of malignant cells preliminary excision for frozen section has been performed at University Hospital with a rigid regard for technique. The tumor is excised with a liberal margin of grossly normal tissue and if the radical procedure is to be carried out the wound is sealed as thoroughly as possible, the original instruments are discarded, and towels and gloves changed.

During the period concerned in this series, 1923 to 1933, such a procedure was far less common than now and in this series of 150 cases was resorted to only fifteen times. Twelve of these cases have been traced and six of them obtained five-year cures. It is dangerous to attempt to base any conclusions on such a small number of cases, but the evidence suggests that there is little or no increased tendency to dissemination in cases which undergo preliminary excision. In evaluating this rate of 50 per cent five-year cures it is important to recall that during the period concerned the procedure was followed in this clinic in only the doubtful- and hence usually early-cases.

AGE OF THE PATIENT

The cases are divided according to decades and the percentage of five-year cures is calculated for each decade (Table 1). Here, as throughout this paper, a case is considered to be a five-year cure only if living more than five years and apparently cancer-free. A number of other cases lived beyond five years with evidences of recurrence or metastasis. The outstanding case of that nature in this series is still living nearly thirteen years after operation and still shows evidences of recurrence. (See Case B).

TABLE 1

Influence of Age on Malignancy of Tumor and Possibility of Cure

AGE	NO. OF CASES	PERCENTAGE OF 5-YEAR CURES*	AVERAGE DURATION OF LIFE FOR THOSE NOT CURED (MONTHS)**	NO. NOT TRACED
20–29	2	50.0	12.0	0
30–39	20	15.0	30.6	0
40–49	34	29.4	19.1	4
50–59	55	20.0	26.0	1
60–69	25	32.0	20.3	3
70–79	14	50.0	35.0	0
Total..........	150	26.0	25.1	8

* Cases not traced are counted as failures in calculating percentage of five-year cures.

** In calculating average duration of life, postoperative deaths are omitted as in these cases the tumor per se had no influence in the duration of life after operation.

Case B: M. G., a 38 year old, white woman had a radical mastectomy by Dr. A. M. Shipley in May, 1926 for carcinoma of the right breast. Pathological examination showed a well-differentiated adenocarcinoma with no evidence of regional metastasis. Recurrent growth appeared in 1930 in costal cartilages and was treated by excision and radium application. Since that time excision and radiation has been performed six times. The patient is living and in good general health, but still has local malignancy. There is as yet no evidence of distant metastasis.

Because of the tendency of carcinoma in the later age groups to pursue a longer course, the average duration of life is also calculated for each decade in patients who were not cured.

Eliminating the inconsiderable two cases in the first group, one sees fairly well the usual progressive increase in percentage of five-year cures as one passes toward the later age brackets. The progressive rise is marred only by the discrepancy in the fifth decade, which is probably attributable to the small number of cases involved. Analysis of the figures on duration of life in patients not cured, however, fails to disclose the anticipated progressive rise.

DURATION OF SYMPTOMS

An effort was made to determine the relationship between duration of symptoms prior to operation and the presence of regional metastasis and percentage of five-year cures. The results are recorded in Table 2.

One is not particularly impressed by these trite figures which merely reiterate that the incidence of metastasis and failures to cure increase in direct proportion to the duration of symptoms. However, one is distressed to find that in a series made up of more than 75 per cent of private patients, roughly half of them have allowed lumps in the breast to persist longer than six months before operation.

TABLE 2

Influence of Duration of Symptoms on the Incidence of Metastasis and the Possibility of Cure

DURATION OF SYMPTOMS	NO. OF CASES	PERCENTAGE SHOWING AXILLARY METASTASIS	PERCENTAGE OF 5-YEAR CURES
2 months or less	30	46.6	30.0
2 to 6 months	48	58.3	31.2
6 months to 1 year	34	58.8	27.9
Over 1 year	38	73.6	15.5

POSITION OF TUMOR

The position of the tumors in the breast is indicated in Table 3. As is the usual finding, the upper outer quadrant was the site of primary growth in more than half of the cases. Regional metastasis, as demonstrated histologically, occurred in 63.6 per cent of the tumors in the upper outer quadrant. This shows no appreciable difference from the 60 per cent regional metastasis in the entire series, but a marked difference from the 46.6 per cent of regional metastasis seen in the thirty cases in which the primary tumor was found in the inner hemisphere. These thirty cases with tumors of the inner hemisphere, however, despite a notably smaller incidence of axillary metastasis, show only a slightly increased incidence of five-year cures (30 per cent) when compared to the entire series (26 per cent), and one assumes that while axillary metastasis may be delayed, pleural and peritoneal extensions occur just as readily in tumors so situated.

PRESENCE OF REGIONAL METASTASIS

Palpable regional lymph nodes were present in seventy-eight of the cases. Twelve of these, however, showed no histological evidence of metastasis. The reason why enlarged nodes should be present in the absence of metastasis or infection is somewhat obscure, but only two of the twelve instances of palpable benign lymph node enlargement occurred in patients with ulcerated lesions. In the seventy-two cases showing no palpable evidence of lymph node metastasis, growth was demonstrated histologically in axillary nodes twenty-four times. Similar findings are reported by Harrington (2) in his comprehensive review of the material from the Mayo Clinic.

TABLE 3

Distribution of the Masses in the Breast

POSITION OF TUMOR	NO. OF CASES
Upper outer quadrant	78
Central	26
Upper inner quadrant	21
Lower outer quadrant	13
Lower inner quadrant	9
No palpable tumor*	3
Total number	150

* Breast removed for carcinoma demonstrated by frozen section after history of bleeding nipple.

Of the 150 cases ninety showed histologically demonstrable lymph node metastasis. In this group there have been 11 per cent five-year cures and 16.6 per cent five-year survivals. In the sixty cases showing no regional metastasis, there were 48.3 per cent five-year cures and 56.6 per cent five-year survivals.

PRESENCE OF ULCERATION

It has long been recognized that an increased operative hazard and decreased possibility of cure obtain in advanced ulcerated cases. The presence of five ulcerated cases among the ten postoperative deaths in this series raises the question of the advisability of a radical procedure in such cases. Hence, these were analyzed as a separate group, with the following results. There were twenty-one cases in the series in which ulceration had occurred. In all cases every effort was made to minimize infection before operation was undertaken. There were five postoperative deaths in this group, which represented an operative mortality of 24 per cent. On the other side of the ledger, however, three of these women were living and apparently cancer-free nine, eleven and twelve years after operation, and two others of the group lived six and eight years before succumbing to metastasis.

Lewis and Rienhoff (4), in their excellent detailed study of the Johns Hopkins material, submit an interesting case report (see Case C) in support of the thesis that radical procedures are justified in advanced disease.

Case C: A 66 year old woman in 1905 was found to have an advanced ulcerative scirrhous carcinoma with regional metastasis to the neck and axilla. At operation the growth was found to be fixed to the fascia and pectoral muscle. Nevertheless, a meticulous dissection of the supraclavicular fossa and axilla, together with a radical sacrifice of skin, was done. The patient was living and apparently cancer-free in 1931 at the age of 93.

HISTOPATHOLOGICAL PICTURE

It has been frequently demonstrated that no known histological criteria are infallible as prognostic guides. However, in the hands of many competent pathologists a certain degree of parallelism does appear to exist between the degree of "differentiation" and the end results in a given series of cases. Each pathologist seems to modify his criteria of differentiation by his individual cumulative experience, hence it is difficult to derive reliable conclusions from the study of different series in which grading has been accomplished by various pathologists. There seems to be, then, a very real need for a didactic set of criteria which may be applied by any competent pathologist with a reasonable expectation of arriving at the same result as that of a colleague for any given tumor. Such a method of grading, needless to say, must fulfill other requirements, viz., it must be broad enough to embrace all or nearly all breast cancers and, while specific enough to allow a considerable number to fall into the lower and higher grades rather than the middle grade or grades, still must be general enough to correspond with the known discrepancy between histology and prognosis of mammary cancer.

The painstaking and exhaustive work of Haagensen (1) resulted in a method of grading which obtained satisfactory results (Table 4) in his series of cases, appeared to allow of application by any pathologist and in general seemed to answer the need. Frantz (3) reported a series of cases graded by this method, with results comparable to those obtained by Haagensen, but also mentions her indebtedness to Haagensen for checking many of the grades. In an effort to learn whether Haagensen's method is universally applicable, the criteria were applied to the histological material in this series, all of which was available.

All of the sections had been prepared by the same technician and there was an average of three or four sections from each tumor. In order to compare the results in this series with those obtained by Haagensen, the same procedure was followed in calculations, that is, the postoperative deaths and patients not traced were eliminated. The results in this series are reproduced in Table 4 and the results obtained by Haagensen are inserted parenthetically for the purpose of comparison.

A glance at this table is sufficient to determine that the results in this series are discouraging. It can be concluded that the personal factor enters into this method of grading.

TABLE 4

Results of Attempted Microscopic Grading as Compared with Those Obtained by Haagensen

	GRADE I	GRADE II	GRADE III	TOTAL
Number of cases	27 (40)*	90 (66)	14 (48)	131 (154)
5-Year survivors	11 (32)	30 (25)	7 (6)	48 (63)
Percentage of 5-year survivors	40.7 (80)	33.3 (38)	50 (13)	36.6 (41)
Average length of life if dead before 5 years	2.08 (3.4)	1.5 (2.3)	1.7 (1.5)	1.6 (2.0)

* Parenthetical figures are those obtained by Haagensen.

In seeking to determine whether any one of the factors used in grading was consistently inaccurate enough to distort the results, some of the individual factors, which Haagensen felt to be indices of relative benignancy, were reapplied to the series with the results shown below.

(a) Marked adenoid arrangement, a characteristic which places a tumor in grade I and which is the most generally accepted of all criteria of relative benignancy, was present in but seven of the cases, four of which showed histological metastasis. There were no five-year cures in this group and but two five-year survivals. There was one postoperative death. The average duration of life for the remaining six was 4.5 years. This prolonged duration of life in the absence of cure is a definite index of low malignancy, but its value as a prognostic criterion in this series is considerably lessened by the absence of cures.

(b) Four cases showed gelatinous degeneration. No report has ever been received on one of the cases. Of the remaining three, two obtained five-year cures and one died from bacterial endocarditis two years after operation. One gets the impression that this characteristic is a reliable index of relative benignancy. (c) Nine cases exhibited the comedo character. There were two five-year survivals, only one of which was a cure. Seven of them had axillary metastasis at the time of operation. The average duration of life for those not cured was thirty-one months. Bloodgood originally noted the relative benignancy of this tumor, but in these few cases the percentage of metastasis and rate of cure are not impressive. (d) There were three cases with a papillary form. Two of them, showing no regional metastasis, were cured. No word has been received from the other, which showed axillary involvement. This is apparently a hopeful quality. (e) There were eighty-three cases showing less than one mitotic figure per high power field. Of these 54 per cent showed axillary metastasis and 25 per cent were five-year cures. Forty-seven cases showed one or two figures per high power field and in this group there were 21.3 per cent five-year cures and 68 per cent showed metastasis. Twenty cases had more than two mitoses per high power field and in this group there were 30 per cent cures and 70 per cent showing axillary metastasis. While there is a slight increase in tendency to metastasize manifested with the increasing number of mitotic figures, no very remarkable influence appears to be exerted by this characteristic.

Haagensen (1) emphasizes in his report that prognosis based on histology is but a rough approximation and it has been proved many times that the most skillful pathologist dare not predict the outcome in any given case. Lewis and Rienhoff (4) express themselves as having "a feeling of great uncertainty as to whether or not the biological characteristics of a malignant growth can be prognosticated from an objective histological examination." They quote two extreme cases which were felt to be histologically indistinguishable (Bloodgood). Both had the same type of operation. In one the tumor was of long standing and the patient lived many years after operation; in the other the tumor was noticed but three months prior to operation and the patient succumbed to generalized metastasis less than six months after operation. They conclude that in their series, in general, "the very cellular types of tumors were, as a rule, more fulminating and malignant than those less cellular, but attempts to grade the relative malignancy or benignancy to a finer definition proved unsuccessful."

It is concluded that even the relatively reliable histological characteristics occur in such a small percentage of breast cancers that they may be regarded more as specific entities than as characteristics of breast cancer as a class. Thus, gelatinous degeneration occurred in but 2.7 per cent of this series and papillary carcinoma in only 2 per cent.

SUMMARY

1. A series of 150 consecutive primary radical mastectomies for carcinoma of the female breast is presented with a follow-up of 94.6 per cent.
2. The operative results are reported.
3. The prognostic significance of preliminary excision of tumor, age, duration of symptoms, position of tumor, presence of regional metastasis and presence of ulceration are considered. As has been demonstrated many times previously, the presence or absence of histologically demonstrable metastasis is the single factor outranking all others in importance.
4. Some of the commoner histological criteria of supposed prognostic significance are evaluated in relation to this series and it is found that the only characteristics which appear to be helpful occur in such a small percentage of cases as to make them of doubtful value as prognostic factors in the general picture of mammary cancer.

The author wishes to express his appreciation to a large group of busy physicians who have taken some pains to secure much of the follow-up information which made this study possible, and in particular to Dr. Hugh R. Spencer and Dr. Arthur M. Shipley for their generous assistance and criticism.

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ACUTE INVERSION OF THE UTERUS*

Report of Two Cases

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The management of cases of inversion of the uterus is a problem which cannot be worked out adequately in the experience of a single individual, simply because no one observer is likely to see more than a very occasional case. Most of us, therefore, must be guided in practice by the published reports of the experience of others. Of the several hundred cases which have been reported, nearly all refer to only one or two cases, with an occasional report of as many as eight or ten. Many more instances have no doubt occurred, but since one of the chief etiological factors lies in faulty technique, many observers are naturally disinclined to publish their experiences. The incidence of inversion is variously estimated as from one in five thousand to as few as one in two hundred thousand cases. The generally accepted average is one in thirty or forty thousand.

While the number of cases reported in the literature is increasing, the author feels that the incidence of inversion of the uterus will decrease with the gradual disappearance of midwives and the continued emphasis in medical schools and hospitals regarding the proper management of the third stage of labor.

It is not the purpose of this paper to present anything conclusive in reporting these two cases, but simply to add them to the literature and to make some comparison of the treatment of these two cases with that emphasized in other reported cases. Textbooks usually speak of inversion of the uterus as being acute or chronic. Any case which has been inverted for longer than one month is considered to be chronic. Both of the following cases are examples of acute inversion.

The first case is that of a white woman, age twenty-six years. She was a primipara at term and had had adequate prenatal care by a private physician who reported nothing unusual. The patient was admitted to the hospital on the first of September, after having been in labor for five hours. However, her pains at that time were of short duration and occurred every fifteen to twenty minutes. During that day there was nothing unusual about the patient's labor. She became quite tired and was given $\frac{1}{4}$ grain of morphine at 1:00 a.m. the following morning. The cervix was fully dilated at about 3:30 a.m. A normal male child weighing 7 pounds, 2 ounces was born without instrumentation or episiotomy at 4:30 a.m. The position was O.L.A.

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A second-degree tear was immediately repaired. The placenta was expressed by vigorous Credé manipulation and with moderate traction on the cord fifteen minutes later. Up to this point the patient had lost a minimal amount of blood but immediately after expression of the placenta there was very active bleeding.

Examination by the physician in charge revealed a completely inverted uterus. No attempt was made at replacement but a tight pack was inserted into the vagina. The patient was given pituitrin and the bleeding seemed checked. It is interesting here to note that in the following few hours, despite the fact that the uterus was known to be completely inverted, there was a notation made in the nurses' record that the fundus of the uterus was massaged frequently.

Consultation with an obstetrician was had about seven hours later. The forty-eight hours following delivery were marked by persistent bleeding in spite of a thorough vaginal pack. The patient was, of course, given intravenous medication of glucose and salt solution and in addition received 1800 cc. of whole blood in three transfusions. Her general condition was becoming progressively worse and it was felt dangerous to delay operation, since she was failing to respond markedly to the treatment for hemorrhage and was beginning to show signs of shock. With ether anesthesia an attempt was made manually to reduce the inverted uterus but this was found to be impossible on account of the marked constriction of the cervix around the neck of the uterus. The abdomen was opened and the typical funnel of the inverted uterus was seen immediately. Contained within this funnel were the bladder, tubes and ovaries. A panhysterectomy was performed without any serious difficulty. The entire operation required only twenty minutes. During this time the patient received another transfusion as well as additional intravenous glucose. The operator noted that the uterus was already markedly necrotic and degenerated. Subsequent to operation there was considerable drainage and the patient had a rather stormy course for the first week. The abdominal incision became infected but responded to the usual treatment. Thirty-five days postoperative the abdominal incision was completely closed. Within a few days the patient was sitting up and was subsequently discharged in good general condition. Three-and-one-half years have passed since the operation and the patient is quite well.

The second case is also a primipara, thirty-five years of age. As in the first case, the patient had been tended during the prenatal period by her private physician who reported that there had been no untoward findings. She was admitted to the hospital on the calculated day of confinement, which was August 25. The first stage consumed about twelve hours, during which time nothing unusual occurred. About six hours before delivery the patient received 1½ grains of seconal. The second stage of labor lasted approximately one-and-one-half hours and at the time of delivery an episiotomy was performed. There was no instrumentation. The baby was a normal female. The position was O.R.A. Fifteen minutes later the placenta was expressed without difficulty by the Credé method. Those present at the delivery were insistent that this expression was accomplished with rather less than the usual manipulation required and that the uterus was not disturbed from the time of delivery until the time of the Credé maneuver. This was in contrast to the previous case in which there was some difficulty in expressing the placenta and in which considerable force was used. The amount of blood lost was again described as minimal.

While the physician was tying the cord, the intern present noticed that the patient suddenly went into a condition of marked shock. One of the obstetricians entered the delivery room just at that moment and, while the patient was being given glucose and caffeine, made a rapid examination which disclosed that there was a complete inversion of the uterus with the cervix tightly constricting its neck.

The patient's degree of shock became more profound in spite of treatment. Intravenous medication was continued and two hours later an attempt was made manually to reduce the uterus. This was successfully and easily accomplished by grasping the circumference of the cervix with Allis clamps, by means of which a peripheral traction could be exerted. The fundus was then reposited without difficulty and was tightly packed with a gauze roll. During this manipulation the patient was practically unconscious and a transfusion was under way. There was no improvement in her condition and she died about an hour later.

In comparing these two cases the most noticeable difference was that in the first instance there was profuse hemorrhage with little shock, while in the second case there was very small loss of blood with profound shock. In the first instance no attempt was made at manual reposition beyond the first failing effort and the patient was intensively treated before being operated upon forty-eight hours after delivery. In the second case the uterus was replaced while the patient was in a profound degree of shock two hours after delivery.

In the medical schools, students are impressed with the dangerous complication that may result from strenuous traction on the cord or too vigorous Credé manipulation, so much so that one is likely to forget that spontaneous complete inversion of the uterus can occur. Various writers describe the incidence of spontaneous inversion to be from 13 to 50 per cent. The author was surprised to find these estimates so high and would be inclined to feel that if the true facts could always be found, spontaneous inversion would probably be less frequent.

In studying the literature there were two facts that were particularly impressive. To begin with, in earlier days there were numerous operations and maneuvers devised for the reduction of inverted uteri. Of these the best known procedure was the Spinelli operation (1899) and the Küstner operation (1893). However, both of these operations were more particularly applicable to chronic rather than acute cases, and for many years only manual reduction was attempted. Not until 1921 did a new method appear and it was then that Huntington devised abdominal approach with reduction by means of re-inverting the uterus with Allis clamps from above.

The second impression one receives from the literature is that there has been a gradual but decided transition from the early idea of immediate reduction to one which emphasizes rather the treatment of hemorrhage and shock without any manipulation of the inverted uterus and disregards this phase entirely until the patient has responded to treatment or until the case has become one of chronic inversion.

In 1925 W. H. McCullagh studied 233 cases in which he found the mortality to be 30 per cent in the presence of shock and 5 per cent in the absence of shock. He found that the cause of death in the order of frequency was attributable first to shock, second to too early operation, third to hemorrhage, and fourth to sepsis. He, therefore, insisted that immediate treatment should consist only of attention to the patient's shock and proposed that the patient should be tided along until involution was complete (four to twelve weeks), when operation could be performed without any increased difficulty and with very much less danger to the patient. His view is concurred in by other observers, including P. G. Brett, D. N. Barrows and J. D. Hancock. However, the Huntington technique which was proposed in 1921 has gradually taken hold and more observers are favoring his idea of operation within twenty-four hours after delivery, with preliminary treatment of shock and hemorrhage. The operation is very simple and can be done rapidly with minimum danger to the patient, since in itself it is not a shocking procedure.

Huntington is opposed to any manipulation from below for the reason that such manipulations tend to bring on excessive bleeding and increase any shock that might be present. In using this technique he and his coworkers have been able to reduce eight cases, all of which recovered and several of which have since had normal pregnancies and deliveries. This seems to the author a decided advantage when one considers that there have been quite a few cases reported which were allowed to become chronic and then reduced by Spinelli's technique, which at subsequent deliveries had ruptured uteri.

N. F. Miller in 1927 reported the study of subsequent pregnancies following inverted uteri. There were fifty-six cases. He found that those uteri which had been manually reduced recurred in 44 per cent of the series, while not a single case recurred where the reduction had been accomplished by operative means.

The discovery of an inverted uterus at delivery is rather startling and it is almost irresistible to attempt to reduce it manually. Many writers in the last few years have felt that one attempt ought to be made at manual reduction, but the opinion is unanimous that this attempt should not be prolonged nor strenuous and that before any operative measures are taken, the primary thing is to combat hemorrhage and treat shock.

In the two cases reported in this paper, the first patient was adequately treated for hemorrhage. Operative intervention was demanded because of the fact that the protruding uterus was becoming necrotic and the patient was beginning to show signs of increasing shock. It should be mentioned that this patient had been given oxytocics in the routine way. This is a practice which is advised against by most writers because they feel that danger to the fundus of the uterus is increased if the cervix constricts its neck. The fundus would soon become edematous owing to obstructed circulation, would subsequently become infected and finally gangrenous. There has been a case reported by Wood in which he was unable to reduce such a gangrenous fundus as the condition of the patient did not favor operation. After six days the fundus sloughed away and the patient recovered without any interference.

The second of the two cases reported should not be too critically compared with other cases, because the patient at the time of manual reduction was clinically in such a state that it was felt she could not recover, whether reduction was attempted or not. The actual reduction of the inverted uterus was readily accomplished and did not make any apparent difference in the patient's condition.

The best opinion today would indicate that the proper treatment for acute inversion of the uterus is to first administer treatment for shock and hemorrhage, to be followed by Huntington's operation as soon as the condition of the patient permits. Such operations as the Spinelli and Haultain should be reserved for chronic cases. The ultimate answer to the treatment of inversion of the uterus will not lie primarily in any operative technique but rather in improved treatment for shock and hemorrhage.

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NEURONITIS FOLLOWING PREGNANCY*

Report of Case

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Mrs. R. H., age 27, Para 1, was admitted to Washington County Hospital on the evening of November 13 in active labor. Her prenatal course was uneventful except for mild nausea and vomiting during the second and third months. The blood Wassermann was negative. The past history contained nothing of importance and her parents and only brother were living and in good health. The patient made little progress and it became necessary to deliver her by forceps of a living female child, weight 7 pounds, 9½ ounces, on the afternoon of November 15. A left lateral episiotomy was done. There was a moderate postpartum hemorrhage which was shortly controlled and the patient returned to bed. Intravenous fluids in the form of normal salt and glucose were administered. The patient's blood picture following delivery was as follows: Red blood cells 2,750,000, white blood cells 9,000, hemoglobin 38 per cent. The differential was normal. She was immediately given iron by mouth and liver parenterally. During the following week convalescence was entirely normal.

On November 22, seven days after delivery, the patient stated that she was unable to taste her food and the next day complained of numbness and tingling in the face, hands and feet. Two days later there was weakness in the flexor groups of both lower extremities and beginning paralysis of the muscles of the left side of the face. The patient rapidly became entirely helpless and a neurological examination on November 30, fifteen days after delivery, revealed the following:

Marked weakness of all muscles supplied by the seventh nerve, especially in the forehead and about the mouth.

Marked weakness of muscles in the forearms more pronounced on the left.

Complete loss of function of the abdominal muscles.

Paralysis of anterior and posterior thigh groups and marked weakness in anterior and posterior lower leg muscles.

No bowel or bladder disturbance.

Muscle sense lost in both feet, also vibration sense.

Plantar reflexes, knee jerk, Achilles and abdominal reflexes were not obtained.

The triceps reflex was not obtained on the right and the periosteal radial reflex was absent on the left.

Pain sense satisfactory throughout.

Outline of discs clear and pupils reacted normally.

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Her course from this point until about December 18 was more or less unchanged. The temperature remained normal, with the exception of a temporary rise to 101.4°F. on December 5. The pulse averaged around 115 but would sometimes jump to 150 and stay there for some hours, even in sleep. The volume at this time was not good. Respiration on one occasion dropped to 10 and remained there throughout an afternoon and evening. The patient was apprehensive at times but always mentally alert. She complained of rather severe generalized aching of the affected limbs and along the dorsal spine, so that frequent small doses of morphine were required for relief. Her speech was thick and there was difficulty in swallowing solid food.

Laboratory findings during this period of the illness showed nothing of particular interest. The secondary anemia disappeared and on December 15 the hemoglobin was 77 per cent, with 3,900,000 red cells. A lumbar puncture was not done at this time because of the patient's critical condition. The urine was normal.

On the afternoon of December 18, which was the twenty-sixth day since the onset of her illness, the patient had what she later termed a "shaking spell". It lasted about forty minutes and was similar to a chill, except that there was no sensation of being cold. As soon as it was over she said she felt "alive again" and was actually able to slightly raise her knees from the bed. This sequence of events was repeated a second time the next day, but to a lesser extent, and after that there was a slow but very definite improvement in the action of all muscle groups. The numbness and aching gradually subsided and there was a return of vibration and muscle sense in the lower extremities. The facial diplegia has shown the least indication of improvement. A spinal fluid examination done several days before the patient's discharge from the hospital on January 9 showed normal sugar and globulin, and a cell count of 21 lymphocytes. Her hemoglobin was 90 per cent and the red cell count 4,500,000.

Convalescence continued satisfactorily during the next several months and an examination on April 18 disclosed a slight weakness of the extensor groups in both legs as the only remaining sign of her illness. She walks without assistance and carries on her usual household duties. The slight difficulty experienced in walking upstairs is slowly disappearing.

Treatment has consisted chiefly of complete bed rest, sedatives in the form of morphine and one or more of the barbiturates as indicated, and large doses of vitamin B complex. She received iron by mouth and was transfused on December 5 with 500 cc. of blood. Salicylates were also administered and massage was begun as soon as the acute symptoms subsided. Cardiac and respiratory stimulants were used when the occasion demanded.

SUMMARY

Neuronitis is a relatively uncommon disease which has been reported variously under the headings of febrile polyneuritis, acute infective polyneuritis, infective neuronitis, myeloradiculitis, and several other terms. It affects both male and female, and all ages equally. It is thought to be of virus origin. As a rule it follows by from one to four weeks some acute infectious disease which may be extremely mild. The onset is gradual or rapid, as in this case, and afebrile. The patients begin by complaining of peripheral sensory disturbances such as numbness and tingling in the extremities. Almost coincident with this there is motor weakness which rapidly progresses to a complete flaccid paralysis spreading through the lower extremities, the trunk, the upper limbs and face. Facial diplegia is considered by some authorities as almost diagnostic. The sphincters are rarely involved. Soreness and generalized aching is common. The deep reflexes are abolished. The illness is progressive over a period of weeks or months. Convalescence is prolonged but recovery is usually complete. Death results from heart failure or respiratory paralysis and the mortality rate in different series ranges from 2 to 20 per cent. The spinal fluid is characteristic and shows an increased globulin out of all proportion to the low cell count.

The author wishes to acknowledge the valuable assistance of Dr. Irving Spear in the management of this case.

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"A WORKMAN THAT NEEDETH NOT TO BE ASHAMED"*

[*2 Timothy ii. 15*]

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Had this much appreciated invitation come to me ten years ago, I should have approached the subject in a different manner from that to which I feel inclined tonight. I should have searched the scriptures, all the medical essayists and many other authorities in an endeavor to preach you a high-toned sermon. It was customary then to give helpful advice and set forth, as well as one could, the ethics to guide the graduating class in the conduct of a profession that we flattered ourselves was and had been for generations held in rather high esteem. Perhaps we had grown somewhat spoiled.

Lately the medical profession finds itself in a situation in which it never expected to be—somewhat on the defensive. It has rarely in times gone by found it necessary to defend itself. We thought the nature and manner of our work and the results obtained made us immune from the necessity of such action. When the rare occasions arose we thought that while individuals and possibly groups many times richly deserved criticism, any attempt to besmirch the profession as a whole was a remote possibility. It may be that even yet we should remain humble and turn the other cheek, or it may be that it would be more fitting to leave any defense to those outside of the profession and to the vindication of time and events. Again it may be that we have been too humble, too self-effacing, too supine. We and our services have been taken for granted for so many years that to say we are cavalierly treated in many instances is to put it mildly.

It is not unreasonable that in a world which is changing so markedly and rapidly there should also be some alterations in relationship between the doctor and the public. There is not time and this is certainly not the occasion to discuss some of the important problems before the medical profession, such as the costs of medical care and distribution of medical services. However, any solution or attempted solution of these problems will seriously affect you and your futures. You have now come of age, you are enfranchised and will have a voice in these matters which I should think you might find confusing.

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* Address at the Pre-Commencement Exercises of the University of Maryland School of Medicine and College of Physicians and Surgeons. The Lyric, Baltimore, June 2, 1939.

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Without arguing for or against any proposals or plans, I would like to review for you a few of the phases of the present situation, to tell you something of your heritage and to say I still feel that you could have chosen a worse profession. In fact, I believe one of the explanations why newspapers, magazines and book publishers feel they have found a public interest in criticism of doctors is because the profession has been on a rather high pedestal.

It is undoubtedly true that there has been a change in the ethical standards in an uncertain percentage of our membership; in this percentage there are perhaps more elements of a business than a profession or of a trade rather than a calling, as we like to think of our work. This change is the result of many things too complex to discuss in detail but include, in addition to industrialization, the depression of the last ten years and many other factors, a pervasion of parts of our profession with the aggressiveness and financial interest seen so well developed in American business, and on the part of the public a love of luxury that supersedes its obligations for necessities.

As regards the latter equation, it is easy to understand that if patients should feel, as most of them formerly did, a strict obligation to pay in accordance with their ability as evidenced by other expenditures for homes, clothes, automobiles, radios, or even for such luxuries as facial and hair attentions, etc., it would enable the doctor to be more charitable and lenient with those who are really in need of free services. This attitude would aid greatly in the adjustment of the care of the indigent and medically indigent with less cost to the state or government. However, taken at its worst, one cannot feel that the situation is as bad as it is painted by some who have had unfortunate experiences and by some of our own profession who for one reason or another have grown somewhat astigmatic. While one hears and always has heard of oppressive fees, hard heartedness and commercialism, these are not the rule and for every one of these instances there are many in which there is no charge or the fee is fixed on a fair basis, or indeed the amount left largely to the patient to decide. In many localities it is possible for physicians, particularly the specialists, to conduct their affairs on a rather strict business plan. In many more localities and in all localities by some doctors, medicine is practiced on what might in truth be called a gratuity system. The patient is charged a fee, but the transaction ends with him paying what he can conveniently afford.

It is a strange turn of affairs that the medical profession of America should be held up to some degree of defamation in the same decade in which it has made more progress than any in the last half century and in the decade in which it has done more for the public in the way of charity than in any similar time in its history. When the depression first developed, doctors willingly and gladly carried their increased burden of free service and did so for a long period of time. While it is not possible to truthfully say that they are carrying the burden so willingly now after the abuses of many kinds they have been subjected to, they are nevertheless carrying it. Provision is made for the supply of the indigent with food, shelter, clothing and hospitalization, but in many localities there is no provision for paying the doctor. In the State of Maryland there are more thousands of persons, made up of the aged, blind, indigent unemployable, indigent employable and dependent children, who receive wholly or in part from state or government, food, clothing and shelter, than there are persons in any city other than Baltimore and yet no uniform medical care for them has been arranged. It is taken for granted that medical care will be supplied in some haphazard manner and we can take pride in saying that in the main it *is* supplied.

It is estimated that American doctors contribute in free service $1,000,000 a day and this is probably an underestimate. There were at the high tide of prosperity in 1929 about 142,000 physicians in the United States engaged in treating the ill. Of these approximately 121,000 were private practitioners, that is, not on full time salaries, who received in fees a little over $1,000,000,000 or about $9.00 per capita of the population. If the figure of $1,000,000 worth of service per day is approximately correct, it can readily be calculated that about one-fourth of the services were free. In this year of prosperity, that is, 1929, the average net income of doctors does not sound so meager, being in excess of $5,000, but this average was elevated by a small 2 per cent of outstanding financial successes, mostly specialists in large cities who earned high incomes. One-fourth of the doctors had net incomes of less than $2,300, 15 per cent earned less than $1,500 and 4 per cent had deficits, yet there are no records of strikes for higher fees. In the year 1930 general practitioners suffered a reduction in net incomes of 20 per cent. Full figures for later years have not been found but are almost certainly much further reduced. In 1931 the average value of free services rendered by each physician in California amounted to $5,000, in Pennsylvania $4,500, in West Virginia $1,033, and in Colorado $2,920. Yet in the State of Minnesota, which differs in no outstanding manner from some of those mentioned, in 1934 the average family expenditure for automobiles was $150.00, tobacco $67.00, candy $37.00, drinks and chewing gum $34.00, radio and music $25.00 and, least in the list, physicians' fees $24.00.

At the height of prosperity all practicing doctors in the country collected $1,000,000,000 in one year; in 1938, with the depression in its ninth year, American women spent $400,000,000 on cosmetics. In the same year, estimated bookmakers' profits, not income, but profits, exceeded the 1929 gross income of private practitioners by $200,000,000. These figures are quoted to show some of the contributions made by the medical profession while the public has been spending freely in other directions. Naturally, this is not the entire answer for the incomes of the spenders are not known, but at least the doctors do not believe that the bookmakers' profits were from the rich alone nor were all cosmetic and other luxury purchases made by them.

Having entered the medical profession with a considerable amount of idealism—wanting to make a living, yes, but wanting to do it in a profession that includes in its performance kindness, charitableness and general decency—and having had your feelings hurt by the harshness of some magazine article, some biased book or some overheard remark, and possibly resenting your government undertaking a criminal, mind you, not a civil but a criminal action against the chief society of your profession, you want to know what else is there in my heritage, besides donating one-fourth of my time and energies to the poor, in which I may take pride.

If in your early years you have a little spare time on your hands while waiting for patients, you may light your pipe and, having read one of the articles referred to and being in a slightly mordant mood, you may meditate along these lines. You may say, "We are accused of wanting to monopolize things medical and surgical." What is the situation about this? "Well", you say, "we are guilty." We monopolize to a large degree, or would like to, the necessary medical attention in the bringing of babies into the world and in the same way giving what comfort and ease we can when individuals of any age go out of the world. We, with the aid of our various types of assistants, including nurses, technicians and others, monopolize to a large extent the privilege of attending contagious and loathsome diseases. We have quite a corner on the efforts at finding the causes of, cures for or methods of amelioration of such widespread antagonists as smallpox, diphtheria, typhoid fever, pneumonia, measles, scarlet fever, yellow fever and all the list too long to enumerate. We and our valued allies have a large monopoly in sweating over and squinting into microscopes, identifying or attempting to identify, culturing and handling little pets like the infectious agents of the diseases mentioned and many others. This same group monopolizes the privilege of doing all postmortems, of examining offensive and ofttimes infectious speciimens of all types. We are rapidly getting a monoply on the privilege of being on twenty-four hour duty as many days of the year as we can stand, as many years as we can stand. Many of the $1,500 or $2,300 earned by the 15 or 25 per cent of our profession are won in the middle of the night or the wee small hours.

Another accusation is that we attempt by our organization to exercise restraints on certain practices of members of our profession and others, that we really restrain trade. "Yes, guilty on this count, too." There have been constant attempts at the restraining of a trade and the building up of a profession. That has been one of our greatest efforts in all the years of our existence and it will be one of our greatest sorrows if a trade supplants our profession. We have been restraining trade in diplomas from unqualified regular schools as well as those of various cults, in so far as we can, all efforts being made to prevent the diploma mill from starting its wheels again. We have prevented hospitals from being pesthouses and have made them the efficient, well-run institutions they are today with their standards of buildings, materials, equipment and personnel. High standards of intern and resident training have been established to help educate and train the doctors of the future. The trade in impure foods, impure drugs, and pure but misbranded drugs has been restricted. Trade in false claims for drugs, foods, and charlatan remedies has been restrained to the extent that we have been sued at various times for a total of $36,000,000 and have paid one cent in damages. We have checked trade in the exploitation of a large number of pitiful cripples and invalids who would otherwise be preys to unscrupulous advertisements of drugs, measures and even so-called doctors. In some of these restraints there has been cooperation between the medical profession and the government which is now the chief critic of the profession.

We or our national organization are accused of being a trust. "Guilty on this count, too." There is a trust, a great trust, of long standing. There is a trust that one can when hurt or ill obtain the services of a doctor or hospital whether or not one is able to pay, and because of the trust the chances are that the doctor who treats the patient will be reasonably competent since he has been trained by competent men in standardized medical schools and hospitals, and there is a trust that, not interfered with, this standardization will grow better and more widespread.

Having gotten into this line of thought, our young doctor indulges himself further and to calm his wounded feelings rehearses some other accomplishments of his chosen profession. What has it, he asks himself, accomplished in this country with regard to the expectation of life at birth? He finds statistics are difficult to obtain because proper records have not been in existence long enough in most states. The best are in Massachusetts. He finds in that State 150 years ago the expectation of life at birth was 30 to 35 years. By 1930 it had increased 26 years for females and 24 years for males. This, he thinks, seems a worthwhile accomplishment and he forecasts that an accurate survey in 1940 will better these figures. Coming to more recent times he finds that in the State of Maryland in 1906 there were 207 maternity deaths and in 1936, 114; in the same period there was a decrease in infant deaths from 4,323 to 1,841; in tuberculosis, all forms, from 2,610 to 1,427; in diphtheria from 328 deaths to 25, and in typhoid fever from 517 deaths to 27. He finds further that on the word of the Commissioner of Health the year 1938 was the most healthy in the history of Baltimore City. He realizes that improved living conditions and other factors have played their part in some of these improvements, but he knows that most of the credit is due his profession.

He then thinks of pernicious anemia and liver therapy, diabetes and insulin, pellagra and nicotinic acid, various types of infections and sulfanilimide; pneumonia and sulphapyrdine, and the sera. He recalls the great advances in the treatment of venereal diseases, all the accomplishments in the control of tropical disease, the changes in Cuba, Puerto Rico and many other countries and regions, making possible the Panama Canal and countless other achievements in regions formerly disease-infested. And when he has thought of all these he has omitted innumerable other triumphs of preventive and curative medicine and its allied branches; he has given no consideration to anesthesia, antisepsis and asepsis with their immeasurable results. The accomplishments seem overwhelming, the more so when he realizes that all those mentioned, except the control of smallpox, have been brought about in less than a hundred years, many of them in less than fifty years, and many in the past decade or half decade.

Reviewing what may be called the substructure of his chosen calling has so engrossed our young doctor that much time has elapsed and he feels apologies are necessary, but having been inducted into a profession with these achievements and the resultant heritage he wonders what must be his code of conduct.

Our recommendations would include nothing new, nothing spectacular, only the old-fashioned fundamentals, perhaps the more old-fashioned the better. The modern modifications do not seem to serve as well as the long established laws of the great prophets.

In a novel of a few years ago called *Green Light* there occurs the observation that "Civilization is an organism, like a tree. If it ever is required to give a final account of itself, the Great Forester, who cuts it down, will find on its stump hard narrow rings made by eras of drought and struggle to survive, and broad, pulpy rings left by seasons of nourishing rains and easy living, but there will be some kind of ring to testify to every year of that growth!"

I trust the tree of your medical life will have many rings; some of them may be hard and narrow; may the broad and pulpy ones be much more numerous. Your presence here, each possessing one of the most difficult degrees to earn, testifies that the nursery period has been carefully cultivated by the nurserymen on this stage and that already there are rings—shall we call them hard and narrow—indicating determination to grow, to bloom and to bear good fruit. If, now, there is transplantation to proper environment, with sufficient but not too much sunshine, the possibilities are very great.

This simile has perhaps been belabored enough but I should like to carry it on still further. What are some of the blossoms and fruits referred to? In this record we are not concerned with medicine as a science. Whether you become a surgeon, an internist or aurist does not matter, though certain specialties are somewhat aloof from some of the pictures I shall try to portray.

You have chosen an exacting profession. You will find occasional discussions as to the difference between a calling and a call. Perhaps some of the distinctions are hairsplitting, but if you have entered medicine as a profession—I congratulate you; if you have entered it as a business—I commiserate with the true followers of "Apollo, the Healer, of Aesculapius and Hygeia and Panacea."

Stephen Paget in *The Confessio Medici* says, "Practice (of medicine) is science touched with emotion", and if you approach it as a cold business of making a living you will many times have deep regrets.

Further in the same text the author speaks of a young doctor and presents the picture so delightfully I shall quote him at length. He says: "To a young man of good disposition, tired of the preliminary sciences, and of humanity stated in terms of anatomy and physiology to the satisfaction of the examiners, this plunge into the actual flood of lives is a fine experience. Hitherto, he has learned organisms; now, he begins to learn lives. He need not go, like other young men, for that lesson, to the slums; for they come to him, and that thrilling drama, 'How the Poor Live', is played to him, daily, by the entire company, hero and heroine, villain and victim, comic relief, scenic effects, and a great crowd of supers at the back of the stage—undesired babies, weedy little boys and girls, Hooligans, consumptive work-people, unintelligible foreigners, voluble ladies, old folk of diverse temperaments, and many, too many, more comfortable but not more interesting people. It all happens so naturally, with such a quick and sure touch; the reality of the day's work, the primal meaning of the crowd, the clash of hand-to-hand encounter with diseases and injuries, urge him to unexpected uses of himself. Here are the very people of the streets, whom he passes every day, here they are coming to him for help, to him of all men, telling him all about it, how it happened, what it feels like, why they did it; looking to him, right away, for advice and physic. There are no two of them quite alike; and their records, laid before him, range through every intermediate shade from purest white to a nauseating black. He begins to see that he has more to learn than the use of a stethoscope; he must learn lives. The problem of lives exalted, or sunk, or messed away, knocks at his heart. Let other young men write lurid little books, and tear the veil from the obvious, and be proud of that achievement; what are they to him, who entertains daily, as a matter of course, both Hell and Heaven?"

One cannot in a short address even mention all of the attributes for which you should strive. Perhaps one of the first to be considered should be honesty. If one is not innately honest, it will be better for him and for the profession to choose another line of endeavor. This term does not limit itself to matters of money but should include one's relationship with one's patients, with the public, with colleagues and, above all, with one's self. A doctor is constantly in a position of trust. He is at times closer to his patients than their religious advisor and he will have many patients who have no such asset. He is constantly in a position to take advantage of those who place their trust in him. To avoid error or what may be interpreted as error he must "lean over backward". It is easy to understand why a doctor should be entirely honest, for the fundamentals of his profession require that his daily efforts be directed toward doing away with conditions that provide him with a livelihood. In debates on methods of medical care it seems to be forgotten that doctors are constantly, and always have been, trying to do away with disease.

If I were talking to a group who had been in practice twenty-five years, I would not feel it necessary to mention the attribute of sympathy. Without meaning to be unkind, I believe the young, not having had so much experience, are less inclined to be sympathetic than those older who have had more trials and seen and felt more suffering and hardship. As you are sympathetic so will you be solicitous, kind and charitable. I do not urge maudlin sympathy for all with whom you come in contact. At times it is necessary to be stern and even harsh. On occasion your sympathy must skip over the patient to the wife or child or some other member of the family who is bearing the brunt of the burden more than the patient. In spite of what has been said and no matter what the future holds, charity is a feature of medical practice that will long be associated with the high type doctor. It is not likely that any reform in medical plans will entirely eliminate this phase of practice and perhaps it will be better so, it may be the chief line between a profession and a business. "Charity" says Ruskin, "is the summit of justice, it is the temple of which justice is the foundation."

One could go on and on adding the attributes of the good doctor, understanding, patience, tolerance, kindness, gentleness and, most important of all, the one that seems to be getting pushed into the background—keep it in your foreground—integrity. But there, I said I was not going to preach you a sermon!

What of this tree of the doctor's life—are there, you ask, no really pretty flowers on it, ever? Some have been mentioned. There are many others, but perhaps the loveliest are seen on other trees in the orchard of his activities. There is a splendid one named courage, of them all this one will thrill you most. Perhaps it is the more apparent because we see a great deal of its antithesis—cowardice and spinelessness—but when you see the fortitude of people under all kinds of vicissitudes, when a mother with superb strength nurses her son month after month, sees him gradually fade away, and can go into his room with a smile and a joke up to the last, and at the end can maintain composure in her deep, quiet grief, we must lift our hats in admiration. It was recently my sad privilege to see a household of gentle people with the father, the breadwinner, paralyzed and bedridden, a daughter in another room dying of nephritis, the mother, who was nursing both, not having left the house for a year, even to see a second daughter who had a major operation performed. This was the entire family—yet all could smile. It is in these and similar instances that one so deeply regrets more cannot be done. If I may supplant a word in a felicitous phrase I would say that, "If we cannot stop disease, it is something to shoe him with felt and prevent his steps from creaking" any more harshly than we can help.

Yes, courage is a lovely flower.

There are many other blossoms. We think of the wonderful friendships; who can pay sufficient tribute to these? Someone has said they are past all words. In the life of a doctor, perhaps, they are closer than in the average and in many instances more numerous.

Dr. John Brown in *Rab and His Friends* said he knew every one in Edinburgh except a few newcomers. Apparently most of them were his friends. They brought him of their treasure, figuratively and literally, too, for there was not a rare engraving, a copy of any old edition, a valuable autograph, anything that anyone in Edinburgh greatly prized, but sooner or later it found its way to Rutland Street, "just that Dr. Brown might see it".

I hurry along, omitting many important branches to come to a cluster of blossoms that belong no more to medicine than to any other profession or business but give endless pleasure to countless people—books. On page 27 in *A Club of One* the author states "A wise doctor should be wise in everything. He should know men—all sorts of men. In questions of race he should see behind and before—progenitors and inheritors alike. Philosophies and religions should be familiar to him. Books and their makers should be the objects of his affectionate regard."

The above is a high ambition, hardly possible in these complex times. One cannot be wise in everything, but a judicious division of one's time will, without sacrificing attention to medicine, permit a wide range of general reading to be done and, too, all culture does not have to come by reading, the conversation of properly selected associates, lectures and even the radio may be sources of learning.

According to Maxim XV in the *Art of Worldly Wisdom* by Gracian "You thus obtain the fame of an oracle by other's toil. Such ministering spirits, distil the best books and serve up the quintessence of wisdom. But he that cannot have sages in service should have them for friends."

The same wise man in Maxim XI said, "Let friendly intercourse be a school of knowledge and culture be taught through conversation."

Rabelais, one of our profession, said, "Nature, I am persuaded, did not without cause frame our ears open, putting thereto no gates at all, nor shutting them up with any manner of inclosures as she hath done upon the tongue, the eyes, and other such out jutting parts of the body. The cause, as I imagine is to the end that every day and every night, and that continually we may be ready to hear and by a perpetual hearing apt to learn."

Those who have the appetite for knowledge and are blessed with good memories will glean learning from many sources, many times unconsciously. On page 205 in *A Club of One* is stated "There is a character in Dickens or somewhere in fiction, whose occupation was in the wine cellar amongst the butts and pipes; he never drank anything, but he was always comfortable by absorption."

It is hoped that pointing out some of the disagreeable phases of your profession will not discourage you, that mention of its rewards will cheer you and that calling to your attention the heritage that is yours will be a comfort to you. May you and your contemporaries and those who graduate in the succeeding years justify some future author in saying, with Robert Louis Stevenson in *Preface to Underwoods*:

"He (the physician) is the flower (such as it is) of our civilization; and when that stage of man is done with, and only to be marvelled at in history, he will be thought to have shared as little as any in the defects of the period, and most notably exhibited the virtues of the race. Generosity he has, such as is possible to those who practice an art, never to those who drive a trade; discretion, tested by a hundred secrets; tact, tried in a thousand embarrassments; and what are more important, Heraclean cheerfulness and courage. So that he brings air and cheer into the sick room, and often enough, though not as often as he wishes, brings healing."

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THE NINETY-NINTH PROGRAM MEETING

The Ninety-Ninth Program Meeting was held on Tuesday, May 16, 1939 in Chemical Hall. The following papers were read:

I. "Fluorinated Aromatic Mercurials and Their Antiseptic Properties" by M. F. W. Dunker, Ph.D. and E. B. Starkey, Ph.D., Department of Chemistry, School of Pharmacy, University of Maryland.

II. "The Secretion Process of the Thyroid in Colored Lantern Slides" by Eduard Uhlenhuth, Ph.D. and Raymond K. Thompson, B.S., Department of Gross Anatomy, School of Medicine, University of Maryland.

Abstracts of these papers appear below.

FLUORINATED AROMATIC MERCURIALS AND THEIR ANTISEPTIC PROPERTIES*†

Melvin F. W. Dunker, Ph.D. and E. B. Starkey, Ph.D.

A brief review of the pharmacological and bacteriological studies reported in the literature was presented. Five new mercurials were prepared and studied. They are: the ortho, meta and para fluorophenylmercuric chlorides, 4-fluoro-2-acetoxymercuriphenol and 4-fluoro-3-chloromercuribenzoic acid. The compounds were prepared by either one or both of two methods, namely, the replacement of the diazonium group by mercury as previously reported by the authors or by the general procedure of direct mercuration. The three isomeric fluorophenylmercuric chlorides prepared had melting points which were higher than those of the corresponding chloro compounds.

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* From the Department of Chemistry, School of Pharmacy, University of Maryland.

† Read at the meeting of the University of Maryland Biological Society, May 16, 1939.

The five fluoromercurials listed above were tested for their antiseptic activity in both water and serum against *Staphylococcus aureus* and *Eberthella typhi.* They were compared with 4-chlorophenylmercuric chloride to obtain an indication of the effect of the fluorine atom on the ring, and with metaphen and merthiolate to gain some idea of their effectiveness. In water it was found that 4-chlorophenylmercuric chloride was slightly more effective than the fluoromercurials, while in serum it was less effective (except for the 4-fluoro-3-chloromercuribenzoic acid which was the least effective compound under all conditions). p-Fluoro-2-acetoxymercuriphenol could be dissolved in either water or serum to the extent of 1 to 1000, whereas the other fluoromercurials were used in suspension. p-Fluoro-phenylmercuric chloride was slightly more effective against Staphylococcus aureus in either water or serum than merthiolate or metaphen but less effective than merthiolate against Eberthella typhi and more effective than metaphen.

THE SECRETION PROCESS OF THE THYROID IN COLORED LANTERN SLIDES*†

Eduard Uhlenhuth, Ph.D. and Raymond K. Thompson, B.S.

A series of Kodachrome lantern slides, 3¼ by 4¼ inches, are shown illustrating the successive structural changes which take place in the thyroid gland of the California newt during a period of uninterrupted daily injection with thyroactivator. These changes show definite relationship to the release and storage of the intrafollicular colloid and exhibit cyclic repetition concomitant with the cycles of the colloid content as demonstrated in quantitative studies of the colloid level of the thyroid gland. Structurally three complete successive cycles have been observed and studied so far.

Each cycle begins with the appearance of large quantities of fluid in the cells and represents a proteolytic enzyme (thyroidax), which when excreted into the follicle breaks down and dissolves the intrafollicular colloid in preparation to its absorption by the cells. At the end of each cycle swarms of small intracellular colloid droplets appear in the cells.

In addition to these keystone stages slides are shown which illustrate the liquefaction and excretion of the droplets, the release of the colloid from the basal cell ends into the blood circulation, the liquefaction and breakdown of the intrafollicular colloid, the degeneration and casting off of cells damaged by forced activity, the plasma granules, and plasma vacuoles and their part in the secretory processes.

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* From the Department of Gross Anatomy, School of Medicine, University of Maryland.

† Read at the meeting of the University of Maryland Biological Society, May 16, 1939.

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PRE-COMMENCEMENT EXERCISES

JUNE 2, 1939

The Lyric, Baltimore

ORDER OF EXERCISES

I. THE PROCESSION: *The Coronation March*, Meyerbeer; *Pomp and Circumstance*, Elgar.

II. INVOCATION: THE VERY REVEREND JOSEPH A. ELLS, Pastor of the Church of the Ascension, Halethorpe.

III. GREETING: H. C. BYRD, B.S., LL.D., SC.D., *President, University of Maryland.*

IV. THE ADDRESS: WALTER D. WISE, M.D., *Professor of Surgery, University of Maryland.*

V. INTRODUCTION OF MEDICAL COUNCIL: By J. M. H. ROWLAND, M.D., SC.D., LL.D., *Dean, School of Medicine.*

ARTHUR M. SHIPLEY, M.D., SC.D., *Professor of Surgery*
J. M. H. ROWLAND, M.D., SCD., LL.D., *Dean of the Faculty*
HUGH R. SPENCER, M.D., *Professor of Pathology*
H. BOYD WYLIE, M.D., *Professor of Biological Chemistry*
CARL L. DAVIS, M.D., *Professor of Anatomy*
MAURICE C. PINCOFFS, B.S., M.D., *Professor of Medicine*
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EDUARD UHLENHUTH, PH.D., *Professor of Anatomy*
CLYDE A. CLAPP, M.D., *Professor of Ophthalmology*
JOHN C. KRANTZ, JR., PH.D., *Professor of Pharmacology*

Walter D. Wise, M.D., *Professor of Surgery*
J. Mason Hundley, Jr., M.A., M.D., *Professor of Gynecology*
William R. Amberson, Ph.D., *Professor of Physiology*
Louis H. Douglass, M.D., *Professor of Obstetrics*

VI. Introduction of Graduates and the Award of Honors:
Dr. J. M. H. Rowland, assisted by Dr. H. Boyd Wylie.

Class Roll

Herman Herbert Baylus
Harry McBrine Beck
Edgar Frank Berman
Aaron Bernstein
Albion Older Bernstein
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Max Ralph Bloom
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William Harvey Leitch
Stephen Lee Magness
John Robinson Magruder
Irving Lowell Marks
William James McClafferty, Jr.
Francis Joseph McLaughlin
Alvin Francis Meyer
Irving Miller
William Shepherd Miller
John Anthony Moran
Charles Hunter Moricle
James Baker Nuttall
David Waugh Palmer
Seigle Wilson Parks
Walter Joseph Pijanowski
Samuel Pillar
Melvin Frank Polek
Dexter LeRoy Reimann
Samuel Rochberg
Edwin Russell Ruzicka
Max Samuel Sadove
Joseph Edwin Schenthal
Isadore Scher
Thomas Scott Sexton
Claude Porter Sherman
Maurice Bert Siegel
Philip Laurens Smoak
Sylvan Daniel Solarz
Herbert Spiegel
William Joseph Steger
Leland Bates Stevens
George Tartikoff
Ramsay Berry Thomas
Wilbur Clyde Thomas
John Peter Urlock, Jr.
Leonard Wallenstein
Jesse Rosenberger Wanner, Jr.
Fuller Barnard Whitworth
Milton Jay Wilder
Sol Wilner
Thomas Luther Worsley, Jr.
Daniel Leonard Zalis

Honors

James Baker Nuttall—*University Prize Gold Medal*

Certificates of Honor

Francis Joseph McLaughlin
William Harvey Leitch
Leland Bates Stevens
Raymond Murray Cunningham
Thomas Scott Sexton
Ramsay Berry Thomas

The Dr. A. Bradley Gaither Memorial Prize of $25.00—Leonard Wallenstein

VII. ADMINISTERING OF HIPPOCRATIC OATH: DR. CHARLES REID EDWARDS.

VIII. BENEDICTION: THE VERY REVEREND JOSEPH A. ELLS, Pastor of the Church of the Ascension, Halethorpe.

IX. THE RECESSION: *March of the Priests*, Mendelssohn.

THE MARSHALLS

Chief Marshall, T. B. AYCOCK, B.S., M.D.

HARRY C. HULL, M.D.	FRANK K. MORRIS, A.B., M.D.
HARRY V. LANGELUTTIG, A.B., M.D.	JOHN E. SAVAGE, B.S., M.D.

COMMENCEMENT EXERCISES

JUNE 3, 1939

Ritchie Coliseum, College Park

COMMENCEMENT PROGRAM

CONCERT—MARCH—*Master Craftsmen*—Huffer.

OVERTURE—*Rosamunde*—Schubert.

SELECTION—*Echoes from the Metropolitan Opera House*—Tobani.

WALTZ—*Danube Waves*—Ivanovici.

EXCERPTS—*Robin Hood*—deKoven.

INTERMEZZO—*Cinderella's Bridal Procession*—Dicken (The University of Maryland Band).

OTTO SIEBENEICHEN, *Director*

THE ACADEMIC PROCESSION—PROCESSIONAL MARCH—*Coronation "The Prophet"*—Meyerbeer (The University of Maryland Band).

INVOCATION—THE RIGHT REVEREND JOHN LAWRENCE SHERIDAN, President, Mount Saint Mary's College, Emmitsburg, Md.

MUSIC—
(a) *Let My Song Fill Your Heart*—Charles.
(b) *Maryland, My Maryland*—Randall. MISS LOUISE BAER, *Soloist.*

ADDRESS—HIS EXCELLENCY, HERBERT R. O'CONOR, Governor of Maryland.

CONFERRING OF HONORARY DEGREES AND HONORARY CERTIFICATES—PRESIDENT H. C. BYRD.

MUSIC—*America*—Audience.

CONFERRING OF DEGREES IN COURSE AND PRESENTATION OF DIPLOMAS—PRESIDENT H. C. BYRD.

MUSIC—*The Star Spangled Banner*—Audience.

Benediction—The Right Reverend John Lawrence Sheridan.

The Academic Recession—Recessional March—*Festival*—Cadman (The University of Maryland Band).

ADDRESS BY GOVERNOR HERBERT R. O'CONOR

In his address to the 1939 graduating class of the University of Maryland, Governor Herbert R. O'Conor warned against "those who will try to win you over to their subversive beliefs in their efforts to promote their disloyal program." He urged the graduates to keep alive "a spirit of sane, independent thinking," telling them that it "can be one of the most potent forces for good in your whole life." The Governor said further that this "is particularly true in this day and age when old standards of life and thought have suffered rude upsets and the whole world knows not what to expect from the future.

"With the broad policy upon which Maryland has always acted firmly entrenched in your mind, you will be able more quickly to detect and to reject these alien doctrines. Never forget that Maryland first gave to the world the principle of universal toleration, and what a boon it proved to be!

". . . Take away with you today, along with your diploma, the full appreciation of the fact that you were educated in Maryland. . . . Let it stand for something important in your life, something of transcending importance, that you have inherited a tradition of education and of thought that is none too prevalent in the world today."

The Governor told the class to keep alive this tradition, stating that it can be preserved to posterity only as it is guarded zealously and put into everyday practice by the "now relatively few on the face of the earth who still hold to the principle that man has been given certain inalienable rights, among them the right to pursuit of life, liberty and happiness."

The Governor declared the reports that "business and professional fields are already overcrowded" were not well founded. He told them instead that unlimited opportunities await thinking men and women.

"Fifty years ago the head of the Patent Office in Washington," he said, "was reported as being ready to resign his post because he felt that business in his department was due for a sharp decline. In the light of what has since transpired, you know that fifty years ago the surface of science's amazing possibilities had barely been scratched. . . .

"In science, in the professions, in business, despite the tremendous advances along many lines which have been made in the last decade, the ultimate has not been attained. Far from it! There is room for advancement in specialized fields, where true progress is yet to be made, where new laws of nature are yet to be discovered, where new progress is yet to be attained as a result of research and experimentation."

DEGREE CONFERRED UPON DR. FRED W. RANKIN

An honorary degree of Doctor of Laws (Honoris Causa) was conferred upon Dr. Fred Wharton Rankin, U. of Md., 1909, author, distinguished surgeon and eminent leader in surgical thought and practice. Dr. Rankin was at one time intern and resident at University Hospital and is one of the best known and best loved of the medical alumni of the University of Maryland. He was presented for the degree by Dr. Arthur M. Shipley at the Commencement Exercises held at College Park.

REUNIONS

CLASS OF 1909 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The Class of 1909 held its thirty-year reunion with an attendance of twenty-five members. On May 31, 1939 a dinner was held at the Maryland Club, at which those present recounted their experiences and recalled many of the instances of their school days. The outstanding feature of all the talks was loyalty to the Class of 1909.

On the afternoon of June 1 a clinic was held in Gordon Wilson Memorial Hall of the University Hospital by Dr. Fred W. Rankin and Dr. George E. Bennett of this class. The clinic was well attended and drew many favorable comments. In the evening the class attended the banquet of the Medical Alumni Association which was held at the Lord Baltimore Hotel.

Throughout the occasion most of the men expressed the desire that another reunion be booked five years hence. Those present were:

Dr. George E. Bennett, Baltimore, Md.; Dr. Paul Brown, Baltimore, Md.; Dr. Arthur E. Cannon, Converse, S. C.; Dr. Arthur L. Fehsenfeld, Trappe, Md.; Dr. Julian M. Gillespie, Washington, D. C.; Dr. Morris B. Green, Baltimore, Md.; Dr. James F. Magraw, Perryville, Md.; Dr. James W. Meade, Fishing Creek, Md.; Dr. Charles A. Neafie, Pontiac, Mich.; Dr. John S. Norman, Gastonia, N. C.; Dr. John N. Osburn, Los Angeles, Calif.; Dr. William G. Queen, Baltimore, Md.; Dr. Fred W. Rankin, Lexington, Ky.; Dr. John W. Robertson, Onancock, Va.; Dr. Harry M. Robinson, Baltimore, Md.; Dr. John T. Russell, Eastport, Md.; Dr. Reed A. Shankwiler, Detroit, Mich.; Dr. Hugh W. Smeltzer, Greendale, Va.; Dr. Maurice I. Stein, Harrisburg, Pa.; Dr. Claude Smink, Baltimore, Md.; Dr. Charles F. Strosnider, Goldsboro, N. C.; Dr. Fred H. Vinup, Baltimore, Md.; Dr. R. G. Willse, Baltimore, Md.; Dr. Eugene B. Wright, Clarksburg, W. Va.; Dr. William E. Martin, Randallstown, Md.

Six members attended the reunion of the Class of 1909 of the Baltimore Medical College. No private functions were arranged but these alumni were present at the clinic held by Dr. Rankin and Dr. Bennett, and later attended the banquet of the Medical Alumni Association.

CLASS OF 1909 OF THE COLLEGE OF PHYSICIANS AND SURGEONS

Twelve of the original forty-five members of the Class of 1909 held their thirty-year reunion on June 1 and 2, 1939. After attending the joint clinic held in Gordon Wilson Memorial Hall, a "get-together" was held before the banquet of the Medical Alumni Association. The members assembled again the next morning for their final meeting. The following members attended the reunion:

Dr. Chadbourne A. Andrews, Tampa, Fla.; Dr. Michael A. Abrams, Baltimore, Md.; Dr. J. B. Dodrill, Webster Springs, W. Va.; Dr. DeWitt Faucet, Gadsden, Ala.; Dr. C. D. Gordon, Mt. Arlington, N. J.; Dr. William Greenfeld, Baltimore, Md.; Dr. W. A. Griffith, Berwyn, Md.; Dr. John E. Hardman, Youngstown, Ohio; Dr. Oliver S. Lloyd, Baltimore, Md.; Dr. A. A. Parker, Pocomoke City, Md.; Dr. William G. Phillips, Skiatook, Okla.; Dr. J. H. Weller, Bridgewater, Mass.

CLASS OF 1914 OF THE COLLEGE OF PHYSICIANS AND SURGEONS

The twenty-fifth reunion of the Class of 1914 of the College of Physicians and Surgeons was held on June 2, 1939 and proved one of the most successful and spontaneously enthusiastic gatherings ever held. Late in 1938 the first letters went out to the members and after a number of follow-ups thirty acceptances were received from a possible forty-seven graduates of the class. Eight of the members had passed away, six were practicing in Puerto Rico and three could not be traced. Twenty-five members of the class came to the reunion, four of the others were ill, and Rae Cather was in Barberton, Ohio, where the unfortunate school explosion prevented his leaving.

Morning and afternoon clinical sessions were held at Mercy Hospital by Doctors Blake, Cotton, Harry Friedenwald, Julius Friedenwald, Edgar Friedenwald, Gardner, Rytina and Samuels. The Sisters of Mercy tendered a luncheon to the visitors, many of whom were former interns and residents of Mercy Hospital. In the evening a "get-together" dinner was held at the Lord Baltimore Hotel, to which the instructors of the Class of 1914 were invited as honorary guests. Twenty-five former members of the Faculty were present. The affair was a great success, with a renewal of acquaintances and friendships which had lasted for many years.

Dr. R. H. Walker of Charleston, West Virginia was toastmaster and almost everyone present was called on for extemporaneous remarks and reminiscences. When Dr. Erwin E. Mayer, chairman of the reunion, read the list of Faculty members and classmates who had passed on through the years, the assemblage rose and in reverent silence drank a toast to their memory.

The committee in charge were in addition to Dr. Erwin E. Mayer, who was still called and remembered as "Kid" Mayer, Dr. Alex Gillis of the Mercy Hospital staff, Dr. R. H. Walker, and Dr. O. H. Bobbitt of Charleston, West Virginia. In view of the great success of the reunion and the spirit of good fellowship which prevailed, it was unanimously decided that the next five-year reunion of the Class of 1914 be held in 1944 in Baltimore.

The following were present:

Class of 1914: Drs. Frank Ayd, Thomas Bess, William P. Black, O. H. Bobbitt, Newell A. Christenson, James D. Crane, George H. Crofton, Alexander J. Gillis, Attie T. Gordon, Homer E. Halferty, Howard C. Heilman, Jesse J. Jenkins, Harry S. Kuhlman, Herbert L. Langer, Harry Lipkin, Joseph Lipskey, John V. McAninch, James P. McManus, Erwin E. Mayer, Stacy T. Noland, Joseph U. Rohr, Harry W. Rosenthal, Bryon W. Steele, R. H. Walker, John B. Webster, James O. Williams.

Faculty: Drs. Cary B. Gamble, Jr., William S. Gardner, Julius Friedenwald, Charles F. Blake, Harvey G. Beck, Albertus Cotton, Abraham Samuels, Caleb W. G. Rohrer, George W. Mitchell, A. Ferdinand Ries, Edgar B. Friedenwald, Lewis J. Rosenthal, T. Fredk. Leitz, Anton G. Rytina, George A. Strauss, Jr., H. K. Fleckenstein, C. C. W. Judd, William Greenfeld, Joseph I. Kemler, Elliott H. Hutchins, Henry T. Collenberg, R. W. Locher.

CLASS OF 1914 OF THE SCHOOL OF MEDICINE, UNIVERSITY OF MARYLAND

The Silver Anniversary Dinner of the Class of 1914 of the School of Medicine, University of Maryland was held on June 2, 1939 at the Lord Baltimore Hotel, Baltimore. An enjoyable program was arranged by the committee in charge under the direction of Dr. Fuller Nance and in addition to the renewal of old acquaintances included a most delightful and varied entertainment. On June 3 the members were invited to a shore party tendered by Dr. E. A. Looper. Those who attended the reunion were:

Dr. Charles C. Ayres, White Hall, Md.; Dr. Yates M. Barber, Gaithersburg, Md.; Dr. Horace W. Byers, Baltimore, Md.; Dr. Arturo Casilli, Elizabeth, N. J.; Dr. Wilford A. H. Councill, Baltimore, Md.; Dr. George B. Crist, Frederick, Md.; Dr. Gilbert L. Dailey, Harrisburg, Pa.; Dr. Walter L. Denny, Jr., Baltimore, Md.; Dr. Richard I. Esslinger, Baltimore, Md.; Dr. Hugh V. Gillson, Paterson, N. J.; Dr. Victor L. Glover, Martinsburg, W. Va.; Dr. George G. Grazier, Hollsopple, Pa.; Dr. Bruce H. Guistwhite, Cumberland, Md.; Dr. Aaron L. Holstein, New York City; Dr. Eugene L. Horger, Columbia, S. C.; Dr. Raymond L. Johnson, Waycross, Ga.; Dr. James W. Katzenberger, Baltimore, Md.; Dr. Morris B. Levin, Baltimore, Md.; Dr. Bayard L. Liggett, Mill Creek, W. Va.; Dr. John F. Lutz, Baltimore, Md.; Dr. Albert D. McFadden, Baltimore, Md.; Dr. Challice H. Metcalfe, Sudlersville, Md.; Dr. Fuller Nance, Baltimore, Md.; Dr. Richard B. Norment, Jr., Hagerstown, Md.; Dr. Marcus Ostro, Baltimore, Md.; Dr. Benjamin Pushkin, Baltimore, Md.; Dr. Abraham Shapiro, Baltimore, Md.; Dr. William M. Stahl, Danbury, Conn.; Dr. Harry M. Stein, Baltimore, Md.; Dr. Charles M. Stephens, Waycross, Ga.; Dr. George L. Timanus, Baltimore, Md; Dr. Raymond M. Troxler, Burlington, N. C.; Dr. Jesse R. Wanner, Salisbury, Md.; Dr. Howard H. Warner, Baltimore, Md.; Dr. William C. Whitesides, York, S. C.; Dr. Frank M. Wilson, Cumberland, Md.; Dr. Austin H. Wood, Baltimore, Md.; Dr. Charles A. Young, Roanoke, Va.

THE 132ND ANNUAL BANQUET

The 132nd Annual Banquet of the Medical Alumni Association of the University of Maryland was held on June 1, 1939 at the Lord Baltimore Hotel, Baltimore. Dr. Charles F. Blake, retiring president of the alumni association, was toastmaster for the occasion and introduced the following speakers:

Dr. H. C. Byrd, President of the University of Maryland
Dr. Charles Conley, Guest Speaker
William H. Kammer, Jr., President of the Senior Class
Dr. J. M. H. Rowland, Dean of the School of Medicine

In addition to the 1939 graduating class, the guests of honor invited were President H. C. Byrd, His Excellency Herbert R. O'Conor, Governor of Maryland, The Honorable Howard W. Jackson, Mayor of Baltimore, Dr. A. J. Lomas, Superintendent of the University Hospital, and members of the Board of Regents.

REGISTRANTS FOR SPRING ACTIVITIES

The following alumni registered during the Spring Activities:

Drs. Harvey S. Brown, Freehold, N. J.; J. W. Campbell, Indiana, Pa.; A. N. Mellott, Ambridge, Pa.; George E. McPherson, Belchertown, Mass.; Howard G. Stevens, New Milford, Conn.; A. E. Dann, Canton, Pa.; Lake Polan, Huntington, W. Va.; Howard Amstrong, Harrisonburg, Va.; J. C. Sleet, Norfolk, Va.; J. F. Flynn, Erie, Pa.; C. E. Imbrie, Butler, Pa.; C. B. Koons, Sipesville, Pa.; John H. Weller, Bridgewater, Mass.; Wm. H. Marsh, Solomons, Md.; P. B. Lonergan, Durant, Iowa; James P. McManus, Bridgeport, Conn.; C. C. Romine, Morgantown, W. Va.; Charles A. Young, Roanoke, Va.; M. Rosen, Philadelphia, Pa.; Raymond L. Johnson, Waycross, Ga.; H. B. McDonnell, College Park, Md.; Edwin R. Raymaley, Wilkinsburg, Pa.; J. Henry Orff, Shillington, Pa.; H. L. Sutton, Newark, N. J.; Joseph Gordon, Ridgewood, N. J.; Goff P. Lilly, New York City; Edwin F. Gouldman, Colonial Beach, Va.; S. J. Sugar, Washington, D. C.; Thomas Bess, Keyser, W. Va.; A. A. Krieger, Pittsburgh, Pa.; Isadore Pachtman, Pittsburgh, Pa.; John B. Webster, New Bedford, Mass.; W. Allen Griffith, Berwyn, Md.; A. Finegold, Pittsburgh, Pa.; Joseph Finegold, Pittsburgh, Pa.; Michael J. Coffey, Newark, N. J.;

Joseph Schwab, Woodhaven, N. Y.; A. W. Valentine, Washington, D. C.; E. L. Horger, Columbia, S. C.; Robert V. Palmer, Palmer, Md.; J. Douglas Crane, Ishpeming, Mich.; F. D. Fortney, Newburg, W. Va.; S. H. O'Neill, Blue Hill, Nebr.; Leroy P. Heck, Mt. Kisco, N. Y.; J. J. Haney, Trenton, N. J.; J. F. Magraw, Perryville, Md.; Herman Cohen, Trenton, N. J.; John R. Bishop, Salisbury, Md.; William J. B. Orr, Washington, D. C.; A. W. Reier, Dundalk, Md.; J. B. Dodrill, Webster Springs, W. Va.; A. A. Parker, Pocomoke City, Md.; C. A. Andrews, Tampa, Fla.; Edward Edelman, Ozone Park, N. Y.; A. Jacoby, Steubenville, Ohio; F. G. Dye, Skaneateles, N. Y.; Lancelot Ely, Somerville, N. J.; G. Orbin Hall, Murrysville, Pa.; E. M. Satulsky, Elizabeth, N. J.; John P. Downes, Newark, Del.; W. C. Deakyne, Smyrna, Del.; H. T. Robinson, Cumberland, Md.; J. L. Van Metre, Charlestown, W. Va.; W. B. Draper, Boonsboro, Md.; C. J. B. Flowers, Harrisburg, Pa.; Morris A. Birely, Thurmont, Md.; M. Franklin Birely, Ridgewood, N. J.; Wm. H. Horan, Manhasset, N. Y. Drs. Wesley J. Ketz, Edgar B. Friedenwald, H. M. Robinson, H. M. Robinson, Jr., J. E. Savage, Milton S. Sacks, Wm. H. Triplett, Cyrus F. Horine, Robert P. Bay, John R. Abercrombie, Milton Solomon, Frank K. Morris, Joseph E. Brumback, Louis H. Douglass, J. Morris Reese, E. Kerr, C. L. Joslin, Franklin B. Anderson, Henry J. Walton, T. N. Carey, C. G. Warner, John E. Legge, Herbert E. Zepp, F. A. Holden, F. A. Pacienza, D. J. Pessagno, Edward A. Looper, Thomas K. Galvin, Edward P. Smith, Monte Edwards, Charles R. Goldsborough, Thomas B. Aycock, J. Morley Hoag, A. Samuels, Kenneth B. Boyd, Carl F. Benson, Harold E. Wright, A. T. Ries, Thomas J. Coonan, Benjamin Rich and Earle M. Wilder of Baltimore, Maryland.

ITEMS

Dr. Arthur M. Shipley, Professor of Surgery, was guest speaker at the meeting of the North Pacific Surgical Association in Spokane, Washington on November 18 and 19, 1938. The titles of his papers were:

"Abdominal Catastrophes Resulting from External Violence"
"Acute Diverticulitis of the Colon"
"The Surgery of the Biliary Duct Apparatus; Its Uncertainties And Anxieties"

On November 17, Dr. Shipley addressed the Spokane County Medical Society on "The Diaphragm as a Surgical Problem". Dr. Aldridge A. Matthews (U. of M. 1900), former superintendent of University Hospital, is President of the Association.

Dr. Shipley resigned as Chief of Surgical Service at Baltimore City Hospitals on January 1, 1939, which position he had held since 1911. He was made Chief Consulting Surgeon and Chairman of the Medical Advisory Board of Baltimore City Hospitals. In May, 1939 the surgical house staff presented a bronze plaque to Dr. Shipley and tendered him a testimonial dinner for his twenty-eight years' service.

During the meeting of the American College of Surgeons held in Baltimore in March, 1939, Dr. Shipley conducted a postgraduate course for physicians and spoke on "Hypertension". He also gave a postgraduate course in respiratory diseases at the American College of Physicians held in March and talked about "Abscess of the Lung".

At the annual meeting of the Medical and Chirurgical Faculty, Dr. Shipley delivered an address on "Appendicitis: Some Observations Based on a Report of 1245 Operated Cases."

The clinics for the Interstate Postgraduate Medical Association of North America were held in the University Hospital on May 8, 1939 and arranged by Dr. Pincoffs, Professor of Medicine, and Dr. Shipley.

On June 15, 1939, Dr. Shipley spoke at the Rural Women's Short Course, University of Maryland Extension Service, College Park, on the subject of "Surgery that a Layman Should Know".

Dr. Shipley was recently elected to the Surgical Research Society.

DR. ROWLAND TO RETIRE AS MEDICAL DEAN

Announcement was made by President Byrd of the retirement this year of Dr. James Marshall Hanna Rowland, Dean of the School of Medicine of the University of Maryland for the past twenty-four years. Upon his retirement Dr. Rowland will assume the title of Dean Emeritus. According to Dr. Byrd, no successor to Dr. Rowland has been named as yet and he will serve as such until an appointment is made sometime before the fall.

Dean Rowland has been associated with the medical school as a student, professor of obstetrics and as dean for more than forty years. Largely through his efforts the relations between the medical schools of his alma mater and of the Johns Hopkins University have grown continually more cordial.

During his long association with the University of Maryland Dr. Rowland has taught more than 3,000 students, who are now practicing medicine in all parts of the country.

Dr. Arthur Karfgin, assistant medical director of the Maryland Casualty Company, has been named chief surgeon of this company. He succeeds Dr. Allen D. Lazenby, vice-president and medical director, who died on April 18, 1939. Dr. Karfgin became assistant surgeon for the Maryland Casualty Company on May 1, 1937 and was made assistant medical director on February 1, 1939.

A native of Baltimore, Dr. Karfgin was graduated from Washington College, Chestertown, Maryland in 1928 and received his medical degree from the University of Maryland in 1932. He served two years' internship at Mercy Hospital and later became resident at the Hospital for Tuberculosis of the Baltimore City Hospitals. For two years he was instructor in physical diagnosis at the School of Medicine, University of Maryland and at the Johns Hopkins Medical School.

Prior to his association with the Maryland Casualty Company, Dr. Karfgin was engaged in general practice in Baltimore.

OBITUARIES

HON. A. W. CHISHOLM, M.D.

Dr. A. W. Chisholm, P. & S. 1894, aged 72, died on May 4, 1939 at his home at Margaree Harbour, Nova Scotia, Canada. Dr. Chisholm received his primary education at Margaree Forks High School, after which he entered St. Francis Xavier University in Antegonish, Nova Scotia. He attended Dalhousie in 1890 and 1891 and then continued the study of medicine at the College of Physicians and Surgeons in Baltimore.

He practiced for a few years at Margaree Forks before moving to Margaree Harbour. Dr. Chisholm represented Inverness County as a liberal member in the House of Commons at Ottawa for eighteen years. Owing to ill health he had given up his practice of medicine three years ago.

J. ARTHUR DOSHER, M.D.

Dr. J. Arthur Dosher, B.M.C. 1903, died in the Brunswick County Hospital, Southport, North Carolina on January 10, 1939.

Dr. Dosher was born in Southport on April 30, 1878. After graduation he returned to his home town to practice. Gradually he devoted more and more of his time to surgery and performed many operations in Southport years before the Brunswick County Hospital was established. During the World War he served overseas with the American Expeditionary Forces and in 1919 was named acting assistant surgeon in the United States Public Health Service at the quarantine station in Southport. He held this post until his retirement in 1937. He also served as county health officer for a number of years and had been chief of the surgical staff at the Brunswick County Hospital since its establishment.

With solemn but simple ceremony a bronze plaque was dedicated on May 12, 1939 to the Brunswick County Hospital in memory of Dr. Dosher. As a further tribute, the State Legislature at its last session passed a bill changing the name of this institution to the J. Arthur Dosher Memorial Hospital.

BENJAMIN HOBSON FRAYSER, M.D.

Dr. Benjamin Hobson Frayser, B.M.C., 1908, U. of Tenn. 1909, aged 49, died at Lexington, Kentucky on March 5, 1937. Dr. Frayser was born in Buchanan, Virginia on October 7, 1887. In 1905, at the age of eighteen, he entered the Baltimore Medical College and after three years he attended a summer session of the University of the South and entered the Tennessee Medical School (now the University of Tennessee Medical School), where he completed his medical course and received his M.D. degree in 1909. While in medical school he was elected an officer in Chi Zeta Chi medical fraternity. He was elected president of his class in his senior year at Tennessee Medical College and president of the general athletic association. Here also he founded Phi Chapter of Chi Zeta Chi. Following academic and medical schooling in the University of Tennessee, Dr. Frayser went to the University of the South as an associate professor in the pharmaceutical department. He received his degree of Ph.G. from the University of the South. Here he founded the national pharmaceutical fraternity, Kappa Phi, and was its Grand Moderator and author of its ritual.

In 1911–12 Dr. Frayser was associate professor of obstetrics and demonstrator of anatomy at Lincoln Memorial University, now the University of Tennessee Medical School.

Dr. Frayser left Lincoln Memorial University to enter government service. The greater part of his professional career was in the medical service of the United States, including the Soldiers' Home Service, the Public Health Service, the Indian Service, the Isthmian Canal Service, the Army, and the Veterans' Administration. He served in fourteen hospitals, in four foreign countries, and three years in the Canal Zone and Santo Tomas Hospital, Panama. As a first lieutenant in the Medical Corps of the Army, he served in Pershing's expedition into Mexico in 1916. At the time of his death he was a major in the Medical Reserve Corps of the Army and had served several tours of active duty which qualified him for promotion to the rank of lieutenant-colonel.

Dr. Frayser was a fellow of the American College of Physicians, frequently attended their conventions and aided materially in the development of the College. He was master and past-president of the Association of Government Surgeons of the United States. His last professional service was rendered efficiently as senior resident physician and surgeon of the Veterans' Administration Hospital at Lexington, Kentucky, where he died.

Dr. Frayser has left much of his personality in several published papers, which show his interest not only in medicine but in fraternal organizations and their history and in religion. He left his entire residual estate of considerable value in trust to be divided equally between the theological department of the University of the South and the Salvation Army.

ALLEN D. LAZENBY, M.D.

Dr. Allen D. Lazenby, U. of Md. 1916, died at Union Memorial Hospital, Baltimore, on April 18, 1939.

Dr. Lazenby was born in Baltimore in 1893. He attended the Baltimore Polytechnic Institute before entering the University of Maryland School of Medicine. After graduation he served as resident surgeon at St. Agnes' Hospital.

From 1918 to 1921, Dr. Lazenby served as a captain in the Medical Corps, United States Army, and was stationed at points in New Mexico and Wyoming. He had been connected with the Maryland Casualty Company since 1921, and was its chief surgeon from 1924 until last January, when he was elected vice-president and appointed medical director.

Recognized as an outstanding authority on the problems of industrial health and occupational diseases, Dr. Lazenby was primarily interested in silicosis and traumatic injuries in industry. He was also active in the Baltimore Chapter of the American Red Cross, served as a member of the executive committee, and formerly was chairman of its first aid and lifesaving committee.

Dr. Lazenby was a fellow of the American College of Surgeons and of the American Medical Association, and a member of the Association of Industrial Physicians and Surgeons. He was chairman of the industrial health committee of the Medical and Chirurgical Faculty of Maryland, and more recently was named a member of the medical committee of the New York World's Fair.

Dr. Lazenby had achieved an enviable position in his chosen field of work. He was a man whose character and ability were acknowledged by the respect and affection of his many friends in the medical profession. His untimely death is greatly regretted.

DR. ROBERT A. WARNER

Dr. Robert A. Warner, U. of Md. 1896, died June 7, 1939, after an illness of two months. He was born and raised within the shadow of the old University at 873 West Lombard Street.

Dr. Warner spent two years as resident at the old Bay View Hospital. During the early part of the century he was health officer of the Western District and continued postgraduate work at Johns Hopkins in neurology until 1911. He was also associated with the late Dr. John C. Hemmeter in the gastro-enterology clinic at the University about this time.

During the World War he served the Government in the United States Public Health Service and afterwards continued with the Veterans Bureau as a full time neurologist and psychiatrist at Fort McHenry.

Dr. Warner was a polished gentleman and a physician of the old school. He had a deep interest in the history of medicine and kept abreast of this, and his specialty, by a regular reading schedule. He was a loyal alumnus of the University and those who knew him miss a staunch, honest friend.

Ashworth, Walter C., Greensboro, N. C.; P. & S., class of 1892; aged 70; died, December 16, 1938, in the Wesley Long Hospital of carcinoma of the bladder.

Barlow, Page D., McMechen, W. Va.; B.M.C., class of 1895; aged 65; died, February 23, 1939, of lobar pneumonia and diabetes mellitus.

Boyles, Joseph Henry, Greensboro, N. C.; P. & S., class of 1903; veteran of the Spanish-American War; aged 65; died, January 24, 1939, of lymphosarcoma.

Brown, James R., Spartanburg, S. C.; B.M.C., class of 1892; aged 72; died, May 25, 1939, of cerebral apoplexy.

Carl, Lenus Albert, Newport, Pa.; B.M.C., class of 1907; aged 60; died, December 1, 1938, in the Harrisburg (Pa.) Hospital of coronary thrombosis.

Colwell, Clifford B., Providence, R. I.; B.M.C., class of 1905; aged 57; died, March 9, 1939, after an illness of two years.

Dick, James McFaddin, Salisbury, Md.; class of 1895; aged 67; died, February 17, 1939, of cerebral hemorrhage and arteriosclerosis.

Duff, Thomas, Wampum, Pa.; B.M.C., class of 1911; aged 56; died, February 16, 1939, of cerebral hemorrhage.

Egbert, Edward Homer, St. Simon Island, Ga.; B.M.C., class of 1905; served during the World War; aged 57; died, February 27, 1939, of coronary thrombosis.

Fahrney, William Edgar, Broadway, Va.; class of 1897; aged 65; died, February 13, 1939, at the University Hospital, Charlottesville, of arteriosclerosis, cerebral thrombosis and pneumonia.

Fetterhoff, Ira Lincoln, class of 1885; aged 74; died, February 25, 1939, of diabetes mellitus.

Gibson, John Shaw, Gibson, N. C.; class of 1905; aged 59; died, January 24, 1939, of coronary occlusion.

Haskins, Thomas Miller, Tulsa, Okla.; P. & S., class of 1886; aged 79; November 8, 1939.

Humphreys, George H., Brockway, Pa.; P. & S., class of 1896; aged 74; died, February 10, 1939, of carcinoma of the liver.

Judkins, John H., Northfield, Vt.; B.M.C., class of 1893; aged 72; died, March 13, 1939, of a heart ailment.

Lindsay, John James, Spartanburg, S. C.; class of 1887; aged 73; died, January 5, 1939, at the Spartanburg General Hospital.

Marston, Charles F., Providence, R. I.; P. & S., class of 1888; aged 74; died, January 5, 1939, in the Rhode Island Hospital of coronary thrombosis and prostatic hypertrophy.

McLean, Burtis W., Jenks, Okla.; P. & S., class of 1906; aged 63; died, January 23, 1939, of injuries received in an automobile accident.

Meyer, Charles H. A., Raspeburg, Md.; B.M.C., class of 1888; aged 78; died, December 1, 1939.

Morrow, Hugh, Patton, Pa.; P. & S., class of 1908; aged 63; died, January 9, 1939, in the Miners' Hospital of Northern Cambria, Spangler, of chronic nephritis.

Murphy, Edward Vincent, Newport, R. I.; P. & S., class of 1899; aged 68; died, March 9, 1939, of cerebral hemorrhage.

Raynor, Russell Wesley, Pikesville, Ky.; class of 1908; aged 52; died, January 20, 1939, of pneumonia.

Reed, John Henry, Logansport, Ind.; class of 1885; aged 78; died, March 2, 1939, of lobar pneumonia.

Riggs, Joseph Potter, Milton, N. J.; class of 1903; aged 59; died, February 24, 1939, in the Franklin (N. J.) Hospital, of lobar pneumonia.

Scanland, John Milton, Imola, Calif.; P. & S., class of 1897; superintendent of the Napa State Hospital; aged 65; died, March 15, 1939.

Seth, Louis Hamilton, Wittman, Md.; class of 1908, aged 55; died, December 25, 1938, of heart disease.

South, Robert B., Rea, Pa.; B.M.C., class of 1896; aged 72; died, November 2, 1938, in the Mercy Hospital, Pittsburgh, of hypersensitive heart disease, nephritis and diabetes mellitus.

Stilley, Jesse Cunningham, Ludlow, Pa.; class of 1912; served during the World War; aged 55; died, November 26, 1938.

Tayntor, Lewis Olds, Conneaut Lake, Pa.; class of 1926; aged 53; died, December 28, 1938, of cerebral hemorrhage.

Trach, David C., Kresgeville, Pa.; P. & S., class of 1891; aged 71; died, January 8, 1939, in the University of Pennsylvania Hospital, Philadelphia.

Walling, Byron W., Poolesville, Md.; P. & S., class of 1875; for several years Mayor; aged 86; died, November 14, 1938, of cerebral hemorrhage and arteriosclerosis.

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Dr. H. Boyd Wylie

Acting Dean, School of Medicine, University of Maryland

BULLETIN
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SCHOOL OF MEDICINE

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Vol. 24 OCTOBER, 1939 No. 2

EDITORIAL

DR. H. BOYD WYLIE APPOINTED ACTING DEAN FOR THE SCHOOL OF MEDICINE

As has been stated in the July issue of the Bulletin, announcement was made by President Byrd of the retirement of Dr. James Marshall Hanna Rowland, Dean of the School of Medicine. Dr. H. Boyd Wylie, Professor of Biochemistry, has been appointed Acting Dean to fill the vacancy thus created.

Dr. Wylie attended Johns Hopkins University from 1905 to 1908, and received his medical degree from the Baltimore Medical College in 1912. He was an assistant in clinical pathology and pharmacology at the School of Medicine, University of Maryland, 1913–14. In 1914–15 he became demonstrator in clinical pathology and pharmacology; associate in biochemistry, pharmacology and clinical pathology, 1915–16; associate professor of biochemistry and pharmacology, 1916–17; associate professor of biochemistry and clinical pathology, 1917–19; professor of biochemistry and head of the department in 1919. This position he has filled with distinction up to the present time. He has been chairman of the Students' Admission Committee since 1936.

During the time Dr. Wylie has been connected with the University his department has grown in value to the institution and has become one of the best organized of any in the American medical schools. Dr. Wylie in every instance has demonstrated his ability to advance with the University in raising it to the high position it now occupies in medical teaching. His positive personality makes him not only admired by the Faculty, but by the student body as well. He may be classed as a teacher of the first rank.

Dr. Wylie is a member of the American Chemical Society, the

Medical and Chirurgical Faculty of Maryland, the American Medical Association and the American Association for the Advancement of Science. He has contributed numerous valuable papers to the medical literature. Among these are "Blood sugar findings in eclampsia and pre-eclampsia" and "Chemical method for detecting pregnancy."

Dr. Wylie's pleasing personality, strength of character and unquestionable ability will warrant the respect and coöperation of his associates and the student body.

CLINICAL STUDY OF THE BONE MARROW IN BLOOD DYSCRASIAS*†

MILTON S. SACKS, B.S., M.D.**

BALTIMORE, MD.

Adequate evaluation of the data obtained through the use of modern hematological procedures frequently necessitates an understanding of the underlying status of the hematopoietic system. Since, in post-natal existence, blood cell formation is largely a function of the bone marrow, examination of specimens of this tissue are often sufficient to supply the key to the solution of certain unusual blood pictures. This fact has been repeatedly demonstrated in recent years in certain hematological disorders in which it has been shown that the peripheral blood does not accurately mirror the condition of the central organs of hematopoiesis. For various technical reasons such as histological complexity, inadequate staining techniques, and relative inaccessibility in vivo, exploitation of the bone marrow from a clinical diagnostic standpoint was comparatively rare. This tissue remained the special domain, largely, of anatomists and pathologists. The introduction within recent years of simpler methods of marrow biopsy and study has resulted in greater clinical interest in the information offered by this procedure. Today, in many hematology clinics, bone marrow study has been added to other routine methods of examination in blood dyscrasias.

ANATOMICAL DATA

Blood cell formation begins in the body stalk and the general connective tissue of the fetus. Sabin (1) has demonstrated that the earliest cells, which are known as megaloblasts and are the first recognizable precursors of the non-nucleated erythrocytes, are differentiated from the lining endothelium of the embryonic blood vessels. The erythrocyte antedates all other types of blood cells in the fetal circulation. Knoll (2) in a study of human embryonic material, demonstrated that granulocytes first appear in the blood stream about two-and-one-half months after the erythrocytes and are then followed by lymphocytes. Only by the third month of fetal life are white blood cells to be seen in appreciable numbers. The liver and spleen soon

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** Hitchcock Fellow in Medicine.

displace the undifferentiated connective tissue as centers of hematopoiesis, but by the fifth month of fetal life are in turn relegated to the background by the bone marrow which now begins to occupy a pre-eminent position as a source of blood cells. At birth it is the most important organ of hematopoiesis and is actively functioning in the medullary cavities of the entire skeleton. For the elucidation of much of the information concerning the embryogenesis of blood cells medicine is greatly indebted to the classical contributions of Sabin (1) and her coworkers. The relationship of bone marrow to hemocytogenesis was first noted by Neumann (3) in 1868.

The distribution and nature of marrow throughout the skeleton at various age periods has been carefully studied by Piney (4), Custer (5), and others. At birth and until approximately the age of seven all marrow has a rich, red, uniform color and is densely cellular. Microscopic fat cells begin to appear at about this time and steadily increase in number until twelve or fourteen years, when macroscopic areas of fat can be seen in the middle of the shaft of the femur. There is a gradual centripetal extension of adipose tissue until in the normal mature adult only small foci of active hematopoiesis are to be found at the proximal ends of the long bones. This process, therefore, produces in the adult a division of marrow into two types, i e., *yellow* and *red*. The latter, which is the actively functioning tissue, is to be found in the short and flat bones of the skeleton and is hence present in the ribs, sternum, clavicles, vertebrae, os innominata, scapulae and skull bones. The existence of large areas of inactive yellow marrow constitutes an important physiological reserve whose potentialities for hematopoiesis are great. Under the influence of certain stimuli, such as chronic hemorrhage or a deficiency of the erythrocyte-maturing-factor (E.M.F.) in pernicious anemia, these areas may again become hyperplastic in a centrifugal manner. In young children the application of similar stimuli frequently results in the formation of extramedullary foci of blood cell formation which retrace the evolutionary pattern of hemocytogenesis above noted.

In the adult the total active marrow has a volume approximately that of the liver (6). Custer (5) studying the relative cellularity of the various medullary cavities demonstrated that the vertebrae and sternum remain densely cellular and display only a small proportion of fat to cells throughout life. Nordenson (7) did differential counts on marrow from vertebrae, the rib, ileum, tibia and sternum and found them essentially similar. Because it is easily accessible and representatively cellular, the latter has become the area of choice for a biopsy sample of the entire marrow. This will be discussed in more detail. Earlier studies on bone marrow by such investigators as Zadek (8) and Peabody (9) were made on the histologically simpler marrow of the tibia. While not representative of marrow activity, the simplicity of structure in the latter area rendered it particularly useful in studying the response to pathological stimuli.

The histological structure of bone marrow presents certain features of great interest. Classical contributions to this subject have been made by Danchakoff (10), Maximow (11), and Doan (12), among others. The adaptation of the vascular pattern in the medullary spaces to hemocytogenesis is worthy of note. The blood supply is usually furnished through a nutrient artery. This medullary vessel after considerable branching terminates in a complex network of relatively wide thin-walled blood vessels called sinusoids, which drain into the longitudinal veins of the marrow cavity. The sinusoids communicate with each other by way of capillaries whose existence has been clearly demonstrated by the work of Doan. He produced depletion and simplification of the bone marrow of pigeons and rabbits by underfeeding and repeated inoculations of typhoid vaccine. The blood vessels of the depleted marrow were injected with India ink and their histological ramifications studied. This work appeared to establish the existence of "closed" circulation in the marrow. The intersinusoidal capillaries being confined within rigid bony walls usually remain only partially patent and constitute a sluggish backwater of the circulation. Histological evidence offered by the work of Doan points to these "erythrogenic" capillaries as the source of red blood cells. The physiological anoxemia in these vessels constitutes an important factor in the normal stimulation of erythrogenesis and perhaps granulocytogenesis (Fig. 1). The continued intravascular origin of red blood cells which is postulated by this interpretation of the histology of the bone marrow is a keystone in the polyphyletic

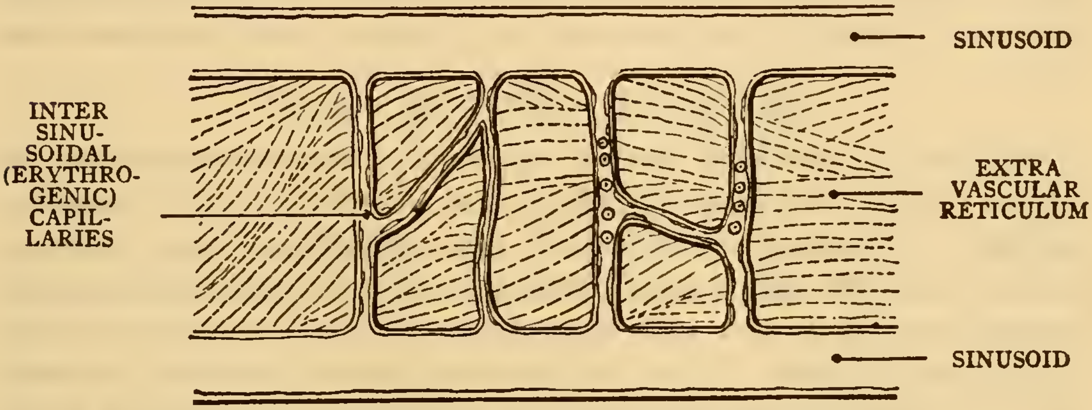

Fig. 1. Diagram of Circulation in Marrow

theory of blood cell formation (Sabin, etc., 13) and as such is the object of considerable controversy among hematologists. The intervascular tissue is in the form of a connective tissue reticulum, associated with which are the reticular cells of mesenchymal origin which retain an embryonic capacity for division and proliferation and are important as the forerunners of the myeloid cells. The fat of bone marrow is in a considerably more labile state than is ordinary adipose tissue. The large expanses of yellow bone marrow composed chiefly of fat cells can be rapidly replaced by active hematopoietic tissue under excessive stimulation. Variations in the fat content of both red and yellow bone marrow is thus a physiological mechanism of great importance in conditions bringing about an increased or decreased demand for blood cells. The existence of nerve endings in bone marrow has never been satisfactorily demonstrated and it is believed that neurogenic stimuli play little or no rôle in hematopoiesis, which is apparently largely influenced by physicochemical changes in the environment.

THEORIES OF BLOOD CELL ORIGIN

As a result of increased clinical interest in bone marrow studies the various theories of blood cell origin have emerged from their relative academic obscurity and assumed new importance and interest. Modern opinions concerning the origin of leukocytes can be conveniently grouped into two categories, i.e., the monophyletic and polyphyletic theories. Even within these broad schools of thought there exist further subdivisions, but the scope of the present paper does not permit more than a brief survey of the outstanding theoretical differences. Hematologists are in agreement that the mother tissue of the blood cells is the reticulo-endothelial system which is to be found in all centers of hematopoiesis. The polyphyletists, whose views are best exemplified in this country by the writings of Sabin (13) and her coworkers, recognize the formation of three distinct types of 'blast cells, through an intermediate stage, a "primitive white blood cell," which are irreversibly committed to the development of a particular series of leukocytes. Distinctions between these 'blast cells are made on the basis of cytological differences observed both in stained smears and in the living supravitally stained cell. However, these distinctions frequently fail to be helpful in the acute leukemias when large numbers of 'blasts can be carefully observed in stained smears. Moreover, the specificity of the cytoplasmic criteria noted in the living stained cell has also been subjected to review and criticism by recent investigators (14). Most hematologists agree concerning the nomenclature and potentialities of the more mature white cells, with the possible exception of the lymphocyte. The continued postnatal development of erythrocytes from the endothelium of the erythrogenic capillaries, which is postulated by the polyphyletists, is likewise a moot question. The validity of these objections to the polyphyletic theory still remains to be immovably established. It is seen that this school of thought distinguishes two parent cells, one an extravascular reticulum cell which is the progenitor of the leukocyte, and the other a specialized endothelial cell from which the erythrocytes are derived.

The monophyletic theory of cell origin is supported by a mass of observations on cell reactions in inflammation and extramedullary hematopoiesis plus the experimental data accumulated through tissue cultures. Among the prominent exponents of this view may be mentioned Maximow (11), Bloom (15), and Downey (16) and his followers. These investigators recognize a single stem cell called the hemocytoblast, which is derived from the R-E system and is the origin of all other formed elements of the blood. The nature of the hemocytoblast is a subject of controversy among the monophyletists. One group, the extreme unitarians, believe it to be identical with the lymphocyte to which they delegate multiple potentialities in regard to further development. Another group, the neo-unitarians, identify the hemocytoblast with the myeloblast in the bone marrow and state that all other cells, red and white, are derived from it under normal circumstances. The latter group, however, recognize the multipotentialities of the lymphocyte, but state that these are not exercised under normal circumstances. In abnormal situations, such as leukemia, this cell can, and has been observed to, produce granulocytes.

The advantages to be accrued by the airing of these complex morphological and physiological problems are definitely appreciable. The elucidation of these questions, which will undoubtedly be aided by continued bone marrow biospy study, must precede the solution of some etiological problems in hematology.

THE MYELOGRAM

The bone marrow is the site of formation of three varieties of cells, namely: those of the granulocytic series, the erythroid elements and the blood platelets. Lymphocytes and monocytes, although present in marrow, are formed largely in lymphoid tissue and the spleen respectively. The determination and tabulation of the varying proportions of adult cells and their precursors in the marrow constitute what is known as the myelogram. Normal values for the myelogram have, until recently, varied somewhat in the literature. The variations have been the result of: (1) differences in technique; (2) variability of nomenclature; (3) the study of an inadequate number of cases. Today, as the result of an increasing experience with bone marrow and standardization of biopsy technique the experienced hematologist can accurately identify better than 95 per cent of all the cells encountered. Mean values for the types of cells normally seen are presented in Table 1. These figures are essentially similar to those of Segerdahl (17), who, in 1935, published the largest series of observations on normal marrow obtained by sternal puncture.

TABLE 1

The Normal Myelogram

	PERCENTAGE
Myeloblasts	1.32
Promyelocyte neutrophils	1.35
Myelocyte neutrophils	15.0
Myelocyte eosinophils	1.37
Metamyelocyte neutrophil	15.69
Staff neutrophils	10.48
Segmented neutrophils	20.86
Segmented eosinophils	1.44
Basophils	0.14
Lymphocytes	16.76
Monocytes	1.67
Erythroblasts	12.88
Normoblasts	
Megakaryocytes	0.03
Histiocytes	0.03
Plasma cells	0.39

Analysis of this table discloses the fact that cells of the granulocytic series constitute approximately 67.6 per cent of the entire group and that nucleated erythrocytes make up approximately 13 per cent. This relationship is frequently expressed as a myeloid-erythroid ratio and deviations from the normal are of clinical significance. It will be noted that approximately 72 per cent of the granulocytes are relatively mature, i.e., ranging from metamyelocytes to polymorphonuclears, and are hence readily available for discharge into the peripheral circulation on demand. This same predominance of mature elements is likewise true for the erythroid series. The existence of this type of cellular distribution in the marrow illustrates another physiological mechanism for the maintenance of a stable level of cells in the peripheral blood. The large numbers of relatively mature cells are readily available for the peripheral blood should occasion arise.

The 'blast cells are rather sharply limited in number and under normal circumstances do not actively take part in the maintenance of mature cells. The latter are usually replenished by division of their own kind. Thus, myelocytes are formed by the division of pre-existing myelocytes. This type of cellular regeneration is known as homoplastic proliferation. Under pathological circumstances cellular proliferation occurs through heteroplastic regeneration, i.e., through the mitotic activity of more primitive cells. With few exceptions the myelogram remains rather constant in pattern throughout life. Kato (18) has recently studied marrow obtained from young infants and has noted that during the first two months of life there is a predominance of lymphocytes.

Disturbances in the myelogram in disease are the result of varied mechanisms, the elucidation of which has been greatly aided by bone marrow biopsy. Among the deviations are recognized such changes as hypoplasia, aplasia, hyperplasia, "maturation arrest," and myelophthisis. The former condition occurs either as the result of undue exposure to such bone marrow toxins as benzol, arsenic, roentgen rays, radium emanations, etc., or "idiopathically." It is observed that frequently one strain of cells may be predominantly involved leading clinically to a leukopenia or thrombocytopenia but eventually displaying itself in its true colors as an aplastic process. Examination of the bone marrow discloses a varied picture. There may be marked reduction in all cell types with a predominance of fat cells which are observed par excellence in trephine biopsies. Or there may be merely a quantitative reduction in the proportion of one type of cells which can be readily demonstrated by a differential count. In some instances of clinical aplastic anemia, Rhoads and Miller (19) have recently demonstrated a marked increase in "primitive lymphoid cells" in the marrow and have suggested that a maturation arrest exists in such circumstances. Doan (20) has recently advocated splenectomy in hypoplastic anemia, the rationale being the elimination of a possible "splenic inhibiting factor."

Marrow hyperplasia occurs in both a controlled and uncontrolled manner. The existence of an anemia for any length of time results in an increase in erythroid cells which can readily be detected in the myelogram. Depending on the etiology of the anemia, the hyperplasia will develop at different levels of erythrocyte development. This phenomenon will be discussed in more detail. Uncontrolled erythroid hyperplasia occurs in polycythemia vera where there is a tremendous increase in the number of normoblasts and at the same time an increase in unripe myeloid elements such as myeloblasts and promyelocytes. Controlled myeloid hyperplasia occurs in response to pyogenic infections. Here the granulocyte percentage rises and there is a slight shift to younger maturation levels. In myeloid leukemia there is an uncontrolled proliferation of very young granulocytes, such as myeloblasts and promyelocytes, which may be so overwhelming as to disturb normal erythrogenesis and platelet formation.

The phenomenon of maturation arrest was first noted as a result of bone marrow biopsy (21). It denotes a paradoxical situation in which there occurs an abundance of precursor cells in the marrow and a diminution or total absence of the mature cells of this series in the peripheral blood. Such a state of affairs exists in agranulocytosis, pernicious anemia, and perhaps other dyscrasias. The application of a specific factor, such as liver extract or pentnucleotide, destroys the "block" and leads to the rapid maturation and delivery of the specific cell to the peripheral blood. The importance of the recognition of this phenomenon lies in its possible application to other blood dyscrasias.

Myelophthisis refers to the invasion of normal bone marrow by tissues foreign to this organ. It may be responsible clinically for anemia, leukopenia and thrombocytopenia or for the production of a leukemoid blood picture with nucleated reds and immature myeloid cells in the peripheral blood. The foreign tissue cells, such as those of Hodgkin's disease, multiple myeloma, and Gaucher's disease, to mention a few, may be observed displacing normal marrow cells in the myelogram.

DESCRIPTION OF MARROW CELLS

Detailed verbal pictures of the immature marrow cells are certainly less effective than adequate illustrations. In discussing these cells at all the author merely wishes to point out certain broad characteristics. In general, the more immature cells can be recognized by the presence of an intensely basophilic cytoplasm usually containing no granules whatsoever. The nucleus is round to oval, usually occupies approximately two-thirds of the cell body, and has a finely reticulated chromatin structure with several definite nucleoli. Minute individual differences between the various 'blast forms are described by the polyphyletists. Even if one admits the existence of a separate 'blast form for each cell series, these differences can only be appreciated by actual observation. In the genealogy of the granulocytes the earliest recognizable form (Fig. 2-a) possesses the characteristics above noted. Maturation is associated with decreasing cytoplasmic basophilia, the acquisition of oxidase-positive specific granules, and changes in

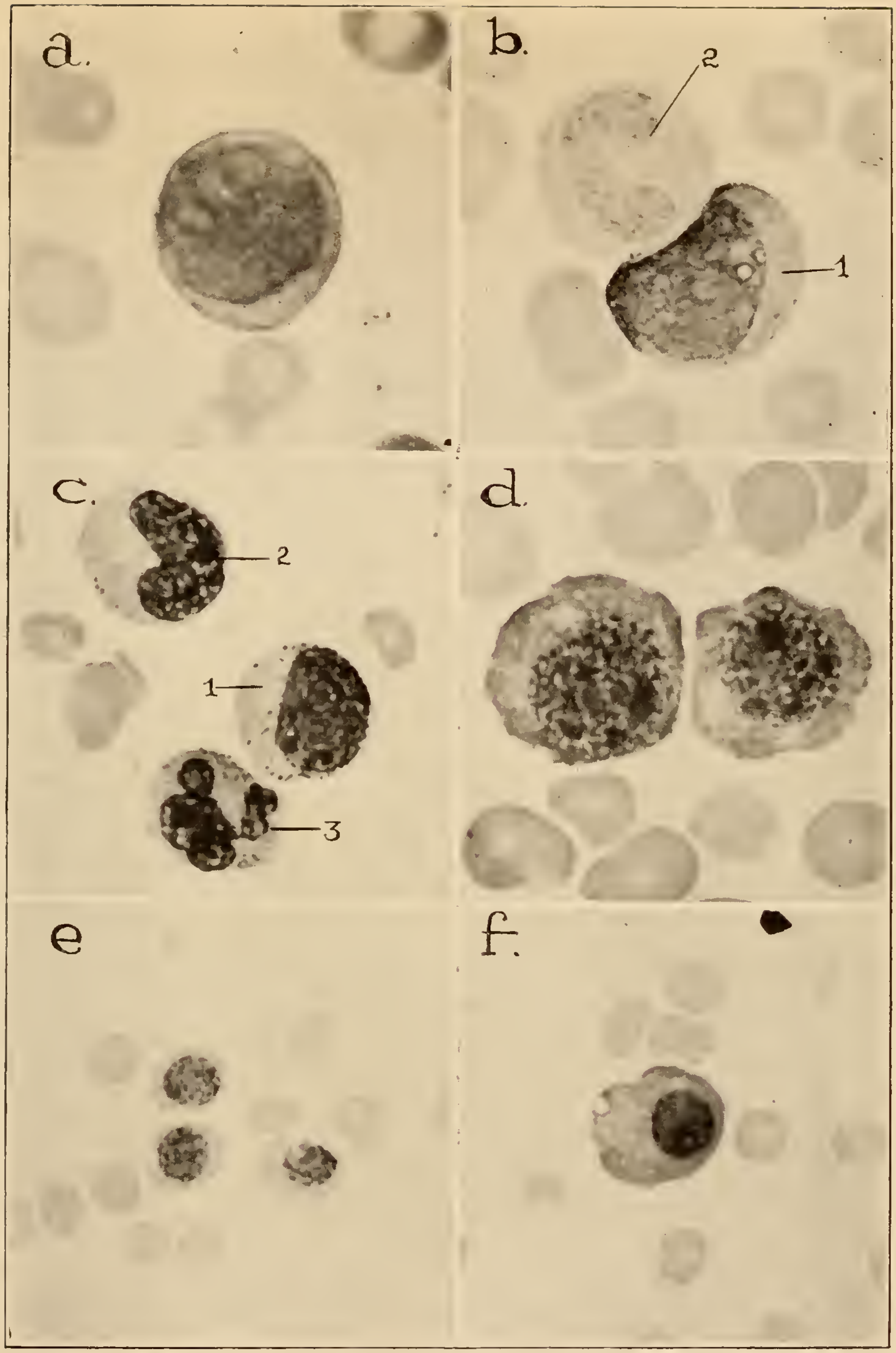

FIG. 2. a. *Myeloblast.* Nucleus occupies approximately two-thirds of entire cell. Cytoplasm nongranular. Pale circumscribed areas in nucleus are nucleoli.

b. *Promyelocyte* (1) Has few granules in cytoplasm. Nucleus similar to myeloblast. *Staff form neutrophil.* (2) Has a nonsegmented nucleus and cytoplasm like that of polymorphonuclear neutrophil.

c. *Myelocyte* (1) Has neutrophilic granules in cytoplasm. Nucleus relatively reduced in size, contains no nucleoli, and is slightly eccentric in position. *Metamyelocyte* (2) is similar to myelocyte but shows beginning reniform nucleus. *Segmented polymorphonuclear* (3).

d. *Megaloblasts.* Cytoplasm is very basophilic. Nuclear chromatin has a characteristic reticulated, openwork pattern.

e. *Normoblast.* Cytoplasm paler and contains hemoglobin. Nuclei more pyknotic.

f. *Plasma cell.* Nucleus is small and eccentric in position. Cytoplasm is intensely basophilic.

the nuclear structure (Fig. 2-b–c). The latter consist in the loss of nucleoli, a coarsening of the chromatin network and progressive alterations in the shape until segmentation is accomplished. The various stages have been named and the terminology is familiar to almost everyone.

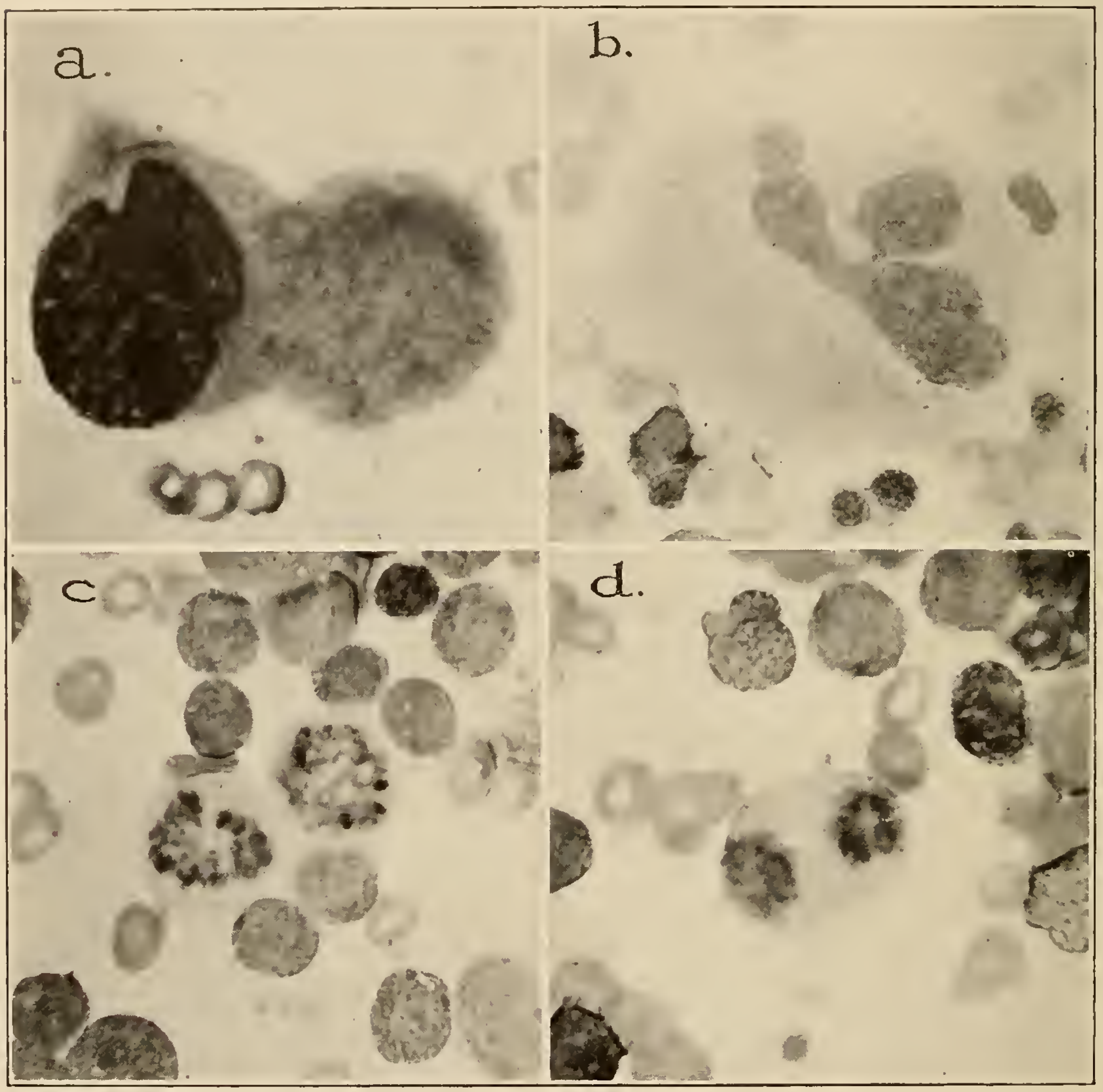

FIG. 3. a and b. *Megakaryocytes.* Size of these cells in relation to those surrounding is evident. Nucleus in (a) less complex in arrangement and suggests an earlier cell.
c and d. *Mitotic figures* in 'blasts. These cells were observed in the marrow of an acute aleukemic leukemia. Mitosis is practically complete in (d).

The successive stages in the development of erythrocytes can likewise be readily detected. The earliest cells are characterized by intense cytoplasmic basophilia. The cytoplasm, however, has a coarser grained structure than that of the myeloblast. The nucleus of the megaloblast (Fig. 2-d) has a delicate scroll-like pattern, resembling a mass of unraveled yarn, which is unmistakable. The nucleus of the normoblast (Fig. 2-e) is similar but of a much coarser pattern. The development of these cells is characterized by the addition of hemoglobin to the cytoplasm, which converts it through a series of blue-gray shades (polychromatophilia) to the intense orange (orthochromia) of the adult erythrocyte. A plethora of names exists in the literature for these varying stages. The simplest classification divides them into megaloblasts, early and late erythroblasts, and normoblasts, in the order of their increasing maturity. The nucleus of the erythroid cell becomes increasingly pyknotic, loses all definite structure, and is eventually either extruded or dissolved.

Certain features of other cells of the bone marrow may be briefly mentioned. The lymphocytes and monocytes resemble their prototypes of the circulating blood. The plasma cell (Fig. 2-f) is encountered occasionally in normal marrow but is present in increased numbers in conditions associated with degenerative changes in the hematopoietic system. Thus, it is to be seen commonly in such clinical entities as aplastic anemia and chronic forms of agranulocytosis. Their function here, as everywhere, is wrapped in mystery. Jordan (22) has recently stated that he believes them to represent abortive efforts by lymphocytes to form erythroblasts. In the author's experience the presence of increased numbers of plasma cells in bone marrow spreads has an ominous significance. Megakaryocytes represent only a small fraction (0.04 per cent) of the myelogram. They are the largest cells of marrow and average 40 microns in size (Fig. 3-a–b). Their cytoplasm has a finely granular appearance and possesses no definite limiting membrane. Clumps of platelets are frequently observed breaking away from the major cytoplasmic mass. The nucleus has a complex convoluted arrangement.

LITERATURE

The introduction of bone marrow biopsy for diagnostic purposes in clinical medicine is of relatively recent origin. In 1908 Ghedini (23) introduced the study of tibial biopsies in leishmaniasis. He also suggested the usefulness of this procedure in the diagnosis of blood dyscrasias. Zadek (8) in 1922, and Peabody (9) in 1927, reported on the characteristic transformation of the marrow in pernicious anemia. The latter's studies were done at a time when liver therapy was in its infancy. He first proposed the concept of a hyperplastic marrow, filled with megaloblasts, which was functionally inefficient, and noted the rapid maturation of cells following the institution of liver therapy. This contribution was of fundamental importance in partially elucidating the pathogenesis of pernicious anemia. However, the fatty, relatively inactive, tibial marrow which was used did not lend itself well for an interpretation of general marrow activity and was soon discarded in favor of the marrow of the sternum. Seyfarth (24) in 1923 first suggested trephination of the latter bone, but it was not until Arinkin (25) in 1927 introduced needle puncture and aspiration of the marrow that this study became widely popular with the medical public. In this country the first report on the clinical study of bone marrow obtained by aspiration was that of Holmes and Broun (26) in 1933. Since that time many papers, which will not be discussed in any detail here, have appeared concerning various phases of bone marrow study.

TECHNIQUE

The two techniques of sternal marrow biopsy in general use today are trephination and needle puncture. The relative merits of these procedures are subjects of current controversy among hematologists. Briefly, trephination is accomplished as follows: an area of the sternum at about the level of the third interspace is prepared aseptically as for any surgical procedure. The skin and underlying tissues are anesthetized with 2 per cent novocain and a longitudinal incision measuring approximately 2 cm. in length is made over the midline of the sternum down to the periosteum. The latter is anesthetized, incised, and elevated, exposing the bare bone. A crown trephine with an internal diameter of approximately 0.5 cm. is introduced by pressure into the bone and rotated manually until the anterior cortex has been perforated. In the adult the cortex has a thickness of approximately 0.5 cm. and the operator frequently experiences a sensation of "give" when the marrow space has been entered. A gentle rocking motion of the instrument loosens the freed button of bone and as the trephine is withdrawn this fragment, to which is attached a portion of marrow, either comes away with it or can be readily picked out. The cavity thus created is curetted with a small mastoid curette and additional fragments of tissue are obtained. This material may suffice, but occasionally another button of bone longitudinally parallel with the first is removed to insure an adequate supply of tissue. The cavity is then filled with bone wax to prevent excessive bleeding and the tissue closed with interrupted sutures. No instances of excessive bleeding or infection have followed this procedure in the author's cases.

For sternal punctures a needle is used which has been prepared in the manner suggested by Young and Osgood (27). An 18 gauge lumbar puncture needle with its stylet in place is shortened to approximately 5 cm. and a new beveled edge created by the use of a carborundum stone. There are today several commercial varieties of sternal puncture needles, but the author has found it unnecessary to employ them. After preliminary cleansing and anesthetization, as above, the needle is introduced at a 70° angle into the anterior cortex of the body of the sternum. Using a combination of direct pressure and a rotating motion one penetrates the anterior cortex readily. Occasionally, here also a sensation of "give" is noted on entering the marrow cavity, but more commonly it is necessary to withdraw the stylet several times and attempt aspiration with the syringe. The latter is of 10 cc. volume and must have a tightly-fitting plunger so that suction may be exerted. Approximately 0.2 cc. of material is aspirated. This substance is of a sanguinous appearance and frequently contains greyish fragments of tissue and occasionally gross globules of fat. The nature of the gross material gives little indication usually of the cytological content.

Coverslip smears are made from the material obtained by sternal puncture. These may be stained by any of the methods used for peripheral blood. Differential counts are done on the basis of no less than 500 to 1000 cells. Some workers aspirate a larger quantity of bone marrow and transfer it to an oxalated tube. This allows for the determination of total nucleated cell counts. Isaacs (28) has established a "normal" figure of 900,000 to 1,000,000 cells /c.mm. The unavoidable dilution of large volumes of marrow with peripheral blood makes these figures somewhat unreliable. The author has not found this determination necessary. The curetted fragments of marrow removed after trephination are likewise utilized for smears. For this purpose the tissue fragments are mixed with a small amount of the patient's serum, which is said to dissolve intercellular material and allows thin smears to be made. Believing that smears distort fine cytological detail, some workers make imprint preparations by gently touching marrow fragments to glass slides. This allows the relationship of cells to one another to remain undisturbed. The activities of living marrow cells may be studied by utilization of the supravital staining procedure. Recently tissue cultures of bone marrow have been prepared and studied by Osgood (29).

The histological procedures adapted to the preparation of sections of bone marrow have been well described by Custer (30) and will not be discussed in detail here. Briefly, the tissue is fixed in Helly's fluid and decalcified in 20 per cent nitric acid. It is then embedded in paraffin and sections cut at a thickness of 4 micra. The necessity of decalcification results in a disturbance of the staining qualities of cells. This is particularly true of the myeloid granules. The author has, therefore, attempted to avoid the decalcification step by removing as much marrow as possible from its bony covering. Sections are best stained by some modification of the Romanowsky stain. Maximow's eosin-azur II, Giemsa, and Wright's blood stain have been used. Tissue sections of bone marrow afford the following observations: (1) arrangement (pattern); (2) activity of one or another cell type (mitoses) (Fig. 3-c–d); (3) hypoplasia of any or all cell types; (4) maturation arrest; (5) areas of focal necrosis; (6) activity of the reticulo-endothelial cells; (7) presence of tumor cells, etc.; (8) evidence of gelatinous degeneration.

The choice of methods of marrow biopsy constitutes a subject of controversy among hematologists today. Trephination, providing as it does material for both smear and section study, probably is more adequate than needle aspiration. However, because it is a minor surgical procedure and should be done in the operating room, and because it necessitates a skin incision with an ensuing scar, the former procedure is less commonly used than the latter. The relative ease of performance, the possibility of securing serial observations on the same patient, the applicability to young children, the convenience of the procedure in regard to ambulatory or dispensary patients, and the very satisfactory cytological preparation, have all conspired to make sternal puncture the more commonly used procedure. A more serious criticism of puncture revolves about the representativeness of the aspiration material. Dameshek, Henstell and Valentine (31) comparing the two methods recently noted that aspiration material contains a lesser number of erythroid elements and that the more primitive erythroid cells are apt to be entirely absent or diminished. This is contrary to the experience of the author. In a recent case of pernicious anemia in relapse he noted as many as 41 per cent megaloblasts. However, more comparative studies of the two procedures are needed before this question will be finally settled. Custer (3), an advocate of trephine biopsy, reports differential counts done on section material. In the author's opinion this procedure is less satisfactory than examination of smears or imprint preparations. Vogel, Erf and Rosenthal (32) reporting recently observations made on 246 marrow punctures state that they are "unable to make accurate differential counts on sections according to the technique of Custer." Sternal puncture has been the first method of choice in studying the marrow of blood dyscrasias. If the results are disappointing from a diagnostic standpoint, trephine biopsy is usually recommended. The latter is an almost essential procedure in suspected cases of aplastic and myelophthisic anemia.

INDICATIONS FOR BIOPSY

Marrow studies in the anemias have thrown new light on the pathogenesis of many of these clinical entities. The condition of the peripheral blood does not accurately mirror the status of the hematopoietic tissue. Thus, a profound anemia may be associated with either hypo- or hyperplasia of the marrow. The hyperplasia may be of a megaloblastic, erythroblastic or normoblastic type. The determination of these factors is of importance both prognostically and therapeutically. As a result of recent studies the clinical concept of a fatty acellular marrow as the background of aplastic anemia has been considerably altered. Rhoads and Miller (19) have recently pointed out that there may be a normocellular or even hypercellular state associated with this clinical picture. They have furthermore demonstrated that there is a marked increase in the percentage of primitive stem cells. On this basis it is postulated that certain cases of refractory, aplastic anemia may be the result of an arrest of erythroid maturation at a very primitive level. Search for a stimulating agent may perhaps lead to a successful therapeutic agent for this disease. Megaloblastic hyperplasia (9) of the marrow has been established as the pathological picture in pernicious anemia and related macrocytic anemia. Arrest of maturation at this level occurs as a result of the absence or deficiency of the erythrocyte-maturing factor (E.M.F.) which is formed by the interaction of an "extrinsic" diet factor with the "intrinsic" factor of Castle (33) secreted in the stomach. This reaction product when supplied clinically in the form of liver extract produces a rapid change in the marrow toward more mature erythroid cells. The observation of these changes places the diagnosis and treatment of the macrocytic anemias on a positive demonstrable basis. The assay of new therapeutic products by their specific effect on human bone marrow has recently been suggested. Specific maturation changes occur days before a reticulocyte peak takes place. The hypochromic anemias, both normocytic and microcytic, have been shown to be associated usually with either erythroblastic or normoblastic hyperplasia in the marrow. At this level of development the stimulus for increased output of erythrocytes is usually iron. Diagnosis of those anemias caused by the crowding out or replacement of erythropoietic tissue, i.e., myelophthisic anemia, may likewise be aided by the finding of increased numbers of abnormal cells in aspiration material. Thus it can be seen that knowledge of the bone marrow picture in a given case of anemia is an aid toward more accurate diagnosis and classification.

Changes in marrow cytology in disturbances of leukocyte production have been extensively studied. In frank leukemia with marked hyperleukocytosis and extreme deviation of the differential count, the marrow merely reflects the picture of the peripheral blood. Hence, in these instances marrow studies are merely confirmatory. However, an aspect of purely academic importance is that which results from the increased opportunities to study the morphology of primitive and abnormal cells. The solution of some of the knotty problems of cell origin discussed above may be aided by this type of investigation. It is in the aleukemic or leukopenic cases of leukemia that bone marrow study is of especial diagnostic help. In these cases, which are seen not infrequently, there may be no lymphadenopathy or splenomegaly and the peripheral blood may show only a small proportion of abnormal cells. Mettier and Purviance (34) in 1937 reported five such cases and commented on the difficulties of accurate diagnosis. The myelogram in these instances shows sufficient alterations in the numbers of the more primitive cells to make the diagnosis more definite. In the clinical entity agranulocytic angina or malignant neutropenia, bone marrow studies have helped elucidate the pathogenesis. With the peripheral blood showing a diminution or even complete absence of polymorphonuclears there exists the paradoxical situation of a marked increase of the more primitive granulocytes in the marrow. This pathological finding has again led to the employment of the concept of a maturation arrest as the explanation of the development of the clinical picture. Myeloblastic or myelocytic increase in the bone marrow constitutes an almost invariable finding here and thus permits this definite entity to be clearly differentiated from other types of leukopenia. It is believed that nucleotide therapy exerts a specific maturing effect on the "dammed-back" stem cells.

Etiological discussions concerning idiopathic thrombocytopenic purpura have often revolved about the question as to whether there is decreased platelet production or increased platelet destruction by the spleen. Studies of the bone marrow in this disease have been made by Lawrence and Knutti (35) and Nickerson and Sunderland (36). The latter in a careful study noted a preponderance of young and adult forms of megakarocytes and suggested that there is actually a functional hyperplasia of these cells resulting from an excessive demand for platelets. This mechanism, therefore, appears to be similar to that which operates in the anemias. These findings have as yet not been adequately confirmed, but should be of considerable importance in distinguishing the idiopathic from the secondary forms of thrombocytopenia. In the former, splenectomy appears to have a definite beneficial therapeutic effect. Sternal marrow biopsy, by demonstrating the status of the hematopoietic tissue, seems to be a useful preoperative diagnostic measure. Its value is illustrated in a case cited below.

Bone marrow studies in sixty-nine cases of polycythemia vera by Reznikoff, Foot and Bethea (37) disclosed the presence of arteriolosclerosis in many instances. This vascular pathology in the marrow was often associated with symptoms of Buerger's disease in the same individual. The authors suggested the possibility of a constant anoxemia in the marrow caused by a diminished blood supply which resulted in hyperplasia and overproduction of the erythroid cells. These findings have not yet been confirmed by other observers. As yet they are only of academic interest but may eventually be of value in differentiating erythremia from the secondary polycythemias which are the result of pulmonary or cardiac disease.

Disturbance of bone marrow pattern by a group of disease entities not specifically involving hematopoietic cells frequently results in the production of bizarre changes in the peripheral blood. These are caused by the appearance of immature myeloid cells and nucleated erythrocytes in a picture otherwise characterized by anemia, leukopenia, and thrombocytopenia. The terms leukemoid or "leukoerythroblastic anemia" have been applied to these pictures. Osteosclerosis, metastatic carcinoma, Hodgkin's disease, multiple myeloma, and the xanthomatoses (Gaucher's, Niemann-Pick's disease and the Schüller-Christian syndrome) are among the entities which produce this upset. The diagnosis of these diseases has been aided by sternal marrow biopsy. Involvement of the marrow, however, is at times of a focal nodular character in these conditions and a single biopsy may not invariably demonstrate the characteristic pathology. Repeated sampling is indicated under such circumstances.

CLINICAL MATERIAL

The author had occasion to perform sternal marrow biopsy, either of the trephine or puncture type, in seventy-two instances which have included a wide variety of blood dyscrasias (Table 2). In these cases the findings have been either of a diagnostic nature or merely confirmatory, and have afforded an opportunity to evaluate under diverse circumstances this relatively new technique. From this material several illustrative cases with comment on the specific value of marrow biopsy in each will be briefly presented.

CASE REPORTS

No. 1. *Pernicious Anemia.* N. H. (Chart #23525), a white female, age 56 years, housewife, was admitted to the service of Dr. L. A. M. Krause at the University Hospital on April 2, 1939, with the complaint of "progressive weakness" since October, 1938. Weakness had become so marked that for three weeks prior to admission the patient had been completely bedridden. Dyspnea and cardiac palpitation were present on slight exertion. The patient complained of "numbness" of the feet for the past year and had also had some "soreness" of the tongue. The past and family histories were noncontributory.

TABLE 2

Clinical Material

	NUMBER OF CASES
Erythrocyte dyscrasias	
Hypochromic anemia	8
Sickle cell anemia	2
Congenital hemolytic jaundice	3
Leuko-erythroblastic anemia	2
Aplastic anemia	3
Pernicious anemia	6
Polycythemia vera	2
Hemorrhagic disease of the newborn	1
Leukocyte dyscrasias	
Myeloid leukemia	
Acute	6
Chronic	7
Lymphoid leukemia	
Acute	2
Chronic	2
Infectious mononucleosis	1
Agranulocytosis	2
Neutropenia	5
Eosinophilia	1
Lymphosarcoma	2
Platelet disorders	
Thrombocytopenic purpura	1
Constitutional thrombopathy	2
Miscellaneous	
Multiple myeloma	2
Hodgkin's disease	2
Mycosis fungoides	1
Splenomegaly (Gaucher?)	1
Subacute bacterial endocarditis	1
Periarteritis nodosa	1
Malaria	1
Typhoid fever	1
Carcinoma of stomach	1
Cardiac failure	1
Dehydration (diarrhea)	1

Physical examination revealed the following significant data: an elderly, gray-haired white female lying comfortably in bed. Her skin had a sallow subicteric tint and there was marked pallor of the mucous membranes. Numerous hemorrhages were seen in the ocular fundi. The sides and tip of the tongue were smooth and somewhat reddened. The mouth was edentulous. No lymphadenopathy, nor any spleno- or hepatomegaly was demonstrable. There was a loss of vibratory sensation from the eleventh thoracic vertebra down. The heel to knee test was poorly performed.

The peripheral blood examination revealed the following data: red blood cells 2,030,000; hemoglobin 39% (5.7 gm. Sahli); white blood cells 4,900; differential normal. The platelets numbered 158,000/c.mm. and the reticulocytes 0.4%. The color index was 0.97; volume index 1.07 (normal 0.85–1.15); mean corpuscular volume 95 cubic micra (normal 82–92). Bleeding and clotting determinations were normal. Gastric analysis displayed complete achlorhydria. The Price-Jones graph indicated macrocytosis.

Sternal puncture was performed on April 5 and revealed that 41.8% of the marrow cells were megaloblasts. The erythroid elements totaled 60.8%, which was indicative of marked hyperplasia. This picture was typical of that of an untreated pernicious anemia.

The patient was started on intensive intramuscular liver therapy and six days later had a peak reticulocytosis of 28.4%. On discharge, May 23, the red count was 4,580,000 with a hemoglobin of 94%.

Comment: In this instance the clinical picture was compatible with pernicious anemia. The color and volume indices were somewhat low. Sternal puncture, however, revealed the typical marrow pathology and offered a definite basis for the institution of intensive liver therapy, with every indication that it would be entirely successful. Thus, six days before a reticulocyte peak the clinician had definitely established the diagnosis. In company with all the other hematological data obtained here, this procedure was only of adjunct importance. However, in the author's opinion it could have easily done away with the necessity of these determinations. Serial observations on such marrows offer an opportunity to evaluate new therapeutic measures, since adequate therapy produces an immediate reduction in the number of megaloblasts.

No. 2. *Aplastic Anemia.* E. D. (Chart #21954), a white male, age 45 years, fisherman, was admitted to the service of Dr. Harry M. Stein at the University Hospital on January 18, 1939, with the complaint of "bleeding of the gums for six months". In July, 1938 the patient began to complain of "soreness of the chest". He was advised to have an extraction of several carious teeth whose gum margins showed oozing of blood. Extraction was followed by persistent bleeding which necessitated cauterization. This, however, was only of temporary value. One month prior to admission the patient first noticed crops of small hemorrhages over the legs, associated with the spontaneous appearance of an occasional larger ecchymotic patch. Increasing weakness was manifested. This was associated with some dizziness. The patient believed he had lost a "small" amount of weight. A review of the systems revealed dyspnea or exertion for several months prior to admission and frequent nausea. Past and family histories were irrelevant.

Examination displayed a well-developed, somewhat undernourished, white male with marked pallor of the skin and mucous membranes. No petechiae or ecchymoses were seen at this time, but a tourniquet test was markedly positive. Persistent oozing of blood from the gums was noted and several areas which had been cauterized with silver nitrate appeared necrotic. Many carious teeth were present. No lymphadenopathy, spleno- or hepatomegaly was present.

A complete blood examination showed: red blood cells 1,200,000; hemoglobin 26% (3.9 gm.); white blood cells 3,850; platelets 34,800/c.mm. The differential count of the white cells was as follows: polymorphonuclear neutrophils 30%; lymphocytes 65%; monocytes 5%. The reticulocytes numbered 4.5%. The bleeding time was four minutes and fifteen seconds; the coagulation time, forty-six minutes. Prothrombin time was fifteen seconds. Other laboratory data were normal except for the continued presence of occult blood in the stools.

Sternal puncture was done on January 20. The aspirated material contained a markedly increased amount of macroscopic fat globules. The myelogram showed a total of only 36% granulocytes of all types. The erythroid elements were slightly hyperplastic, totaling 30.6%, and the lymphocytes were increased to 30%. No megakaryocytes were seen in the marrow. These findings suggested hypoplasia of the marrow with especial reference to the granulocytes and megakaryocytes. Trephine biopsy was subsequently performed which confirmed this impression by virtue of the finding of almost completely fatty sternal marrow.

The patient's course was steadily downhill in spite of all therapy. He received approximately ten liters of blood during his hospital stay. Exitus occurred on March 25, 1939.

Comment: This case is of interest in that it resembled essential thrombocytopenic purpura. Splenectomy was strongly considered, but its futility was adequately demonstrated by determination of the status of the marrow. Preoperative marrow examination seems indicated in all cases considered as candidates for splenectomy.

No. 3. *Agranulocytosis.* A. Z. (Chart #23998), a white female, age 43 years, housewife, was admitted to the service of Dr. L. A. M. Krause at the University Hospital on April 24, 1939, with the complaint of "sore throat and fever". For two weeks prior to admission the patient had frequent headaches, for which she had taken approximately 60 grains of aspirin daily with little relief. On April 17 she took 10 grains of amidopyrine in addition to the aspirin. Five days later she began to complain of sore throat, feverishness and increased headache, and experienced several chills. Nausea and vomiting occurred several times that day. On April 23 she was seen by the family physician who prescribed sulfanilamide for the angina. The patient received 6 gm. of the drug during that day, but since no improvement was apparent hospitalization was advised on the next day.

Examination revealed a very toxic patient whose temperature was 104°F., pulse 120, and respiration 22/minute. The skin was of sallow color and the sclerae had a faint icteric tint. An examination of the buccal cavity showed mild pyorrhea alveolaris, marked edema and congestion of the pharyngeal mucosa extending to the soft palate, uvula, and tonsillar pillars. Small areas of ulceration were observed also, scattered over the buccal mucosa. Marked bilateral cervical adenitis was present. The remainder of the examination was negative.

Shortly after admission a white cell count was performed which showed a total of 600 cells/c.mm. The differential was composed of 3% polymorphonuclear neutrophils, 83% lymphocytes, and 14% monocytes. Throat culture was positive for diplococcus pneumoniae and a gram-positive rod. The following morning a complete blood picture showed practically normal values for all other formed elements. Chemical examination of the blood showed an elevation of bilirubin to 3.4 mg./100 cc.

Sternal puncture was performed on the morning of April 25. No polymorphonuclears were observed in the marrow, but the myelocytes were up to 37.3%. These cells showed "toxic" changes which consisted of cytoplasmic vacuolization, nuclear karyolysis and the presence of large, basophilic granules in the cytoplasm. The lymphocytes were increased to 34%.

In spite of specific therapy which resulted in an increase in segmented polymorphonuclears to 94% and a total white count of 9,800, the patient developed an increasingly severe laryngitis with marked edema which necessitated an emergency tracheotomy. Bilateral, confluent, lobular pneumonia developed and the patient died on April 27. An autopsy was performed.

Comment: This case presented the classical picture of agranulocytosis with maturation arrest at the myelocyte level in bone marrow. The finding distinguishes this clinical entity specifically from other types of neutropenia. Thus, aleukemic leukemia and aplastic anemia have frequently been mistaken for agranulocytosis. Nucleotide therapy, which did produce an increase in peripheral granulocytes, failed owing to the presence of an overwhelming pulmonary infection.

No. 4. *Aleukemic Leukemia.* S. S., a white male, age 19 years, was admitted to the service of Dr. M. C. Pincoffs at Mercy Hospital on October 8, 1938, with the complaint of "chronic ulcer on right leg". This appeared spontaneously on the medial aspect of the lower third of the right leg two months prior to admission. Because of the lack of response to all local medication the attending physician had prescribed sulfanilamide. After several days of this medication a sallow, icteric tint to the skin was noted by the physician. The finding of a marked anemia led to hospitalization.

Examination revealed a well-developed, well-nourished, young white male, who was alert and cooperative. Marked pallor of the skin and mucous membranes was present. Several carious teeth were observed. No lymphadenopathy, spleno- or hepatomegaly was present. A circular ulcer, 3 cm. in diameter, with edges well defined and slightly undermined, and having a slight serous discharge, was present on the right leg.

The blood picture on admission was as follows: red blood cells, 1,380,000; hemoglobin 40%; white blood cells 3,350. The differential was: polymorphonuclear neutrophils 29%; polymorphonuclear eosinophils 1%; juvenile neutrophils 2%; myelocyte neutrophils 7%; promyelocytes 4%; lymphocytes 57%; monocytes 2%. Repeated blood examinations showed a persistence of the anemia and a white count which varied from 2,300 to 7,700. During all this time immature granulocytes in small numbers were always present in the peripheral blood. The platelets were slightly reduced and numbered 267,960/c.mm. Other laboratory data were noncontributory, except for the basal metabolic rate which varied from +10 to +33%.

Sternal puncture was first performed on October 21. At this time there were 18.9% promyelocytes and 14.1% myeloblasts in the marrow. Re-examinations of the marrow were done on November 22, 1938 and February 24,1939. All of these showed a persistent elevation in primitive myeloid cells. These findings led to the definite establishment of the diagnosis as an aleukemic myeloid leukemia of a chronic type. The patient has been followed in the medical clinic since his discharge from the hospital. At the time of writing, the anemia remains at a level of about 1,000,000 and the leukopenia has become even more pronounced. No lymphadenopathy or splenomegaly has developed.

Comment: Disturbances of the peripheral blood produced by sulfanilamide have been the subject of many reports in recent medical literature. These have included cases of hemolytic anemia, agranulocytosis and hyperleukocytosis with immature white cells in the circulation. In the above case such possibilities were of necessity included in the differential diagnosis. Bone marrow studies, however, clearly established the diagnosis in a case which otherwise would have constituted a diagnostic problem. The case falls into the category described by Mettier and Purviance (34) as "aleukemic leukemia without splenomegaly or lymphadenopathy". Anemia is one of the outstanding features. It is of the myelophthisic or replacement type and will not respond to therapy.

CONCLUSIONS

The value of bone marrow studies may be conveniently grouped into several categories. First, in certain instances it is of *diagnostic* value. Thus, in aplastic anemia, aleukemic leukemia and the refractory anemias resulting from bone marrow encroachment by extraneous cellular elements of a widely varied nature, this technique is one of the few fruitful procedures leading to the establishment of a correct diagnosis. Secondly, in a wide variety of blood dyscrasias, marrow study is largely *confirmatory*. This is particularly true in the frank leukemias, agranulocytic angina, pernicious anemia, polycythemia vera, congenital hemolytic jaundice and various hypochromic anemias. In many of these instances bone marrow studies were of considerable aid in elucidating the pathogenesis of the disease. Thirdly, the *investigative* opportunities offered by bone marrow study have only begun to be tapped. New avenues of investigation are opened through the use of bone marrow cultures as suggested by Osgood (29). Problems of cell origin will undoubtedly be aided by this type of investigation. The mutation of cell types can be studied anew and the effects of drugs can be minutely observed in vitro.

From the purely clinical standpoint, one may say that bone marrow study in vivo is certainly not the open sesame to the diagnosis of all the disease entities involving so vast and ubiquitous a tissue as the hematopoietic system. However, the added clarity of understanding of the blood dyscrasias afforded by past studies in marrow structure and the possibilities of future fruitfulness make a knowledge of bone marrow morphology and physiology a distinct asset to the clinician.

The trephine biopsies on cases listed in this paper were performed by Dr. George H. Yeager.

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TREATMENT OF PNEUMOCOCCAL PNEUMONIA WITH SULFAPYRIDINE*†

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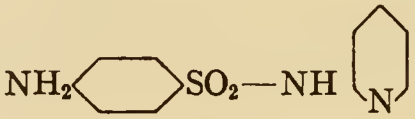
The introduction of any new method of treatment or specific form of therapy has always been accepted with a considerable feeling of doubt by the medical profession until sufficient time has elapsed to prove its real worth. This, however, has not been entirely true in the instance of sulfapyridine, a name adopted by the council of the American Medical Association in January, 1939 for 2-(p-aminobenzenesulfonamido) pyridine or sulfanilamidopyridine. The outstanding results obtained from the start convinced the medical profession of its value.

The drug known as sulfapyridine was synthesized by Ewins and Phillips, who were working in the Dagenham laboratories of May and Baker, Limited, British manufacturing chemists, by replacing one of the hydrogen atoms of the SO_2NH_2 group of sulfanilamide by a basic pyridine group. The resulting compound has the following structural formula:



2-(p-aminobenzenesulfonamido) pyridine

Its trade name in England was M. & B. 693. In the United States the name sulfapyridine has been universally adopted.

Sulfapyridine is a white crystalline, almost tasteless solid, soluble in water at ordinary temperatures to the extent of approximately 1 in 1000.

Important steps were taken by Lionel E. H. Whitby (11) of Manchester, England, who showed that this new compound was effective in mice against pneumococci of types I, II, III, V, VII and VIII, especially against types I, VII and VIII. Shortly thereafter the clinical use of the drug in human pneumococcus infections was instituted in England, with favorable results reported.

The Federal Food, Drug and Cosmetic Act prevented the compound from being placed on the market in the United States until sufficient

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evidence had been gathered to prove its worth clinically. To obtain this information the drug was distributed free by the manufacturers to various hospitals throughout the country for clinical experimentation. Not until March 10, 1939 was sulfapyridine released for general use.

The mechanism of the action of this drug is as yet undetermined and only theories may be presented:

1. Sulfapyridine is bacteriostatic on the invading organism, thus enabling the body defenses to overcome the infection.
2. Toxins are neutralized and phagocytic activity is stimulated.
3. Enzymes, which are essential to the organism from the standpoint of reproduction, nutrition and protection against bodily poisons, are delayed by the drug.

In the University Hospital a supply of sulfapyridine was obtained late in December, 1938 for clinical experimentation. Its use was instituted both on the pediatric and medical wards of the hospital. The results obtained in infants under three years of age are reported by J. E. Bradley and G. J. Wells in another paper appearing in this issue of the Bulletin. The present paper reports observations on thirty-nine adults with pneumococcus pneumonia who were treated with sulfapyridine in this hospital between December 28, 1938 and July 31, 1939.

METHOD OF STUDY

Selection of cases. When sulfapyridine was first available in the University Hospital, only those cases were selected for which type specific antipneumococcus sera could not be obtained. Since early spring, however, the drug has been used in all cases of pneumococcus pneumonia entering the hospital. This report deals solely with the thirty-nine cases in which sulfapyridine was the only specific form of therapy employed.

Diagnostic criteria. In the thirty-nine reported cases the presence of an authentic pneumococcus pneumonia was established by the following criteria: a characteristic history, physical signs of consolidation, presence of a pneumococcus in the sputum or blood, and a roentgenogram confirming the pulmonary involvement. Treatment was not withheld until all of the criteria were fulfilled, but was begun when a tentative diagnosis of pneumococcus pneumonia had been made; moreover, only those cases which ultimately proved to be pneumococcus pneumonia were included in this series.

A typical history was obtained in each case. Thirty-one had a preceding cold, twenty-four a definite chill, pain in the chest was experienced by thirty-three, and bloody sputum was present on admission in thirty-two of the cases.

CHART I

Types of Pneumonia and Deaths—Time Period from December 28, 1938 to July 31, 1939, Inclusive

TYPE	1935–1936		1936–1937		1937–1938		1938–1939*		1938–1939	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
1	11	3	7	2	10	2	6		6	
2	1	1	1		2		1		2	
3	1		2		2	1	1		7	
4	28†	7	1		2				3	
5					3		2			
6							2			
7	2		1		4		1		2	
8			5	2	2				2	
9					1					
10							1			
11					1					
12					1				1	
13							1			
15							1		1	
16							1			
17					1		1			
18					1	1				
19									2	
20									1	
22									1	
27							1	1		
5 & 10									1	
No specific type	18	7	25	7	18		1		9	
Total......	61	18	42	11	48	4	20	1	38	0
Fatality Rate.....	29.5%		26.2%		8.3%		5%		0%	

Typing of sputum was not attempted in one case of this series.

* Cases of present season which were not treated with sulfapyridine alone.

† Includes old type IV.

Physical signs were quite definite in all instances and the following analysis represents those signs found on admission. The percussion note was impaired in twenty-three cases, dull in thirteen, and in three it was flat. The breath sounds were suppressed in thirteen, tubular in eighteen, and bronchial or bronchovesicular in six. Crepitant râles were noted in thirty-one cases.

Pneumococcus was present in either the sputum or blood in every instance, a specific type being found in twenty-nine of the cases (Chart I). The Neufeld reaction as advocated by Sabin was employed, using fresh sputum or punctate material from a lung puncture. If satisfactory typing was not obtained by this method, the material was then injected into the peritoneum of a mouse and also cultured. The latter two procedures are important as six of the cases did not reveal a type until these methods were used.

Roentgenograms were made in thirty-six cases and showed definite evidence of consolidation in thirty-four. The later course of the disease furnished evidence of the development of consolidation in the negative cases or those not roentgenized.

Methods of following progress of cases. Ward patients were seen regularly by the visiting staff and observed carefully several times each day by the house officers.

Blood counts were made at regular intervals, with an average of four to five counts on each patient during his or her stay in the hospital.

The urine was examined at various intervals for any evidence of blood, bile or renal damage.

The amount of free sulfapyridine in the blood was determined on the morning following the first administration of the drug. Subsequent checks were always made at approximately the same time each day, with an average of three to four determinations on each patient during the course of therapy.

METHOD OF ADMINISTRATION OF SULFAPYRIDINE

Oral dosage. The entire thirty-nine cases received the drug orally. The following program was used in the majority of cases of this series:

1. An initial dose of 3 to 4 grams was administered.
2. Thereafter 2 to 3 grams were given every six hours until the temperature remained normal for twenty-four hours.
3. For the succeeding three days the patient received 0.5 to 1 gram every six hours.

Only critically ill patients received the initial dose of 4 grams and a follow-up of 3 grams every six hours.

The average total dosage given per patient was 28.5 grams.

The dosage was modified according to the concentration of sulfapyridine found in the blood. A level of not less than 4 mg. per cent nor more than 10 mg. per cent was taken as an objective. In this series an average concentration of 6.8 mg. per cent was obtained on the first morning after treatment was started and the average maximum concentration during treatment was 9.2 mg. per cent.

Preparation of drug for oral administration. The number of 0.5 gram tablets required for the dose were pulverized and suspended in a solution of sodium bicarbonate, using gram for gram. Attention is called to the fact that sulfapyridine is not very soluble and a considerable amount of the drug is lost unless the glass is well rinsed with water after the original dose is administered. In some few instances milk or fruit juice was used to suspend the drug.

Some observers suggest the use of capsules, jelly or a gastric sedative. E. K. Marshall (10) has prepared a sodium salt of sulfapyridine, not as yet on the market, which can be given intravenously.

CHART II

Age Incidence—Time Period from December 28, 1938 to July 31, 1939, Inclusive

AGE PERIOD	1935-1936		1936-1937		1937-1938		1938-1939*		1938-1939	
	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths	Cases	Deaths
12-19	8	1	4	1	7	1	2		3	
20-29	18	4	12		15		6		9	
30-39	11	3	9	4	12		4		7	
40-49	10	1	3	1	5		7		8	
50-59	8	5	8		4		1	1	5	
60-69	4	2	4	4	2				5	
70-79	1	1	1	1	3	3				
80-89	1	1	1						2	
Total...	61	18	42	11	48	4	20	1	39	0

* Cases of present season which were not treated with sulfapyridine alone.

RESULTS OF THERAPY

Effect on mortality rate. The thirty-nine adult cases here reported all recovered. It is, of course, realized that this series is quite small and that the element of chance cannot be excluded. Moreover, it is possible that the type of disease was not of the average severity. In favor of this supposition is the fact that during this same period there were fourteen other cases of pneumococcus pneumonia treated in this hospital with specific sera alone, with only one death, a result decidedly more favorable than the usual experience with serum treatment. However, in the sulfapyridine group the clinical features of the cases up to the time treatment was instituted did not suggest unusual mildness of the disease. Fifteen of the thirty-nine cases were caused by types I, II or III pneumococci (Chart I); three cases showed bacteremia on admission; nine cases had more than one lobe involved and twelve cases in the series were over fifty years of age (Chart II).

On the whole, it is felt that the favorable results cannot be discounted on the grounds of an unusually mild form of the disease.

In other series of cases of pneumococcus pneumonia treated with sulfapyridine which have been reported from other clinics in recent months, the mortality has ranged between 4 and 8 per cent.

This striking reduction in the mortality rate from this disease is best appreciated if a comparison is made with the mortality previously observed in such cases when not treated with specific remedies and when treated with specific sera.

From a compilation of reports from various parts of the country from 1889 to 1937 (8), a total of 19,540 cases of lobar pneumonia may be grouped. The mortality in this group was 32.8 per cent.

In reported series of cases treated with serum the mortality of type I pneumonia has been decreased from 29 per cent to about 14 per cent, and that of type II infections from 37 per cent to 22.5 per cent. The results from sera in the less common types are not as well known but are approximately in the same proportion. The use of sera therefore nearly halved the mortality.

It has been interesting to compare the mortality rate with sulfapyridine with that observed in the same months in previous years in the University Hospital (Chart I). It will be noted that in 1935–36 and 1936–37 the rates were 29.5 and 26.2 per cent respectively. In these years little use could be made of serum, but in 1937–38 when specific sera were freely available the rate fell to 8.3 per cent and in the sulfapyridine series of 1938–39 to zero.

The results so far obtained in this and other clinics with sulfapyridine constitute, therefore, an important further reduction in the mortality from pneumonia.

EFFECT ON THE CLINICAL AND LABORATORY FEATURES

The average duration of fever from the beginning of treatment was 26.14 hours. The fever was considered ended when the temperature dropped and remained below 100° F. An analysis of the cases shows that the duration of fever after starting treatment was practically the same, no matter on what day of the disease treatment was instituted.

Usually the pulse rate falls with the fever but is a little slower in reaching the normal level.

The respiratory rate likewise usually falls with the fever, but if consolidation occupies a large portion of the lungs, or if there is much abdominal distention, or if pleural effusion is present, there will be only a minimal reduction in the rate of respiration. A drop in tem-

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perature without a fall of the respiratory rate should suggest the possibility of a pulmonary complication.

The cough may prove quite annoying even after a drop in fever, and the sputum may become more abundant. With the fall in temperature there is an improvement in the well-being of the patient and a diminution of the toxemia.

Physical signs. No immediate effect may be expected on the pneumonic process in the lungs in those cases treated with sulfapyridine. There is usually no immediate resolution and the time required is probably about the same as that necessary after a natural crisis. One might say that the duration of the dangerous phase of the disease is diminished but the duration of disability is little changed. The average duration of time in bed was 11.9 days and the patients spent an average of 19.3 days in the hospital.

CHART III

Complications of Pneumonia—Time Period from December 28, 1938 to July 31, 1939, Inclusive

COMPLICATION	1935–1936		1936–1937		1937–1938		1938–1939*		1938–1939	
	Cases	%	Cases	%	Cases	%	Cases	%	Cases	%
Empyema	4	6.6	3	7.1	2	4.2	8	40	1	2.6
Pleural Effusion	4	6.6	4	9.5	4	8.3	1	5	4	10.3
Pericarditis	2	3.3								
Unresolved Pneumonia	2	3.3							1	2.6

* Cases of present season which were not treated with sulfapyridine alone.

Leukocyte count. In the majority of cases the fall in the white blood cell count was somewhat slower than that of the fever and pulse rate. In some patients who were quite ill there was a relatively low count in proportion to the severity of the disease, and in these cases there was an increase in the number of leukocytes after administration of the drug, later followed by the usual fall.

When the leukocyte count does not fall it should suggest the possibility of some complication.

Complications. It cannot be stated as yet whether sulfapyridine decreases the number of complications, but we do know that it does not eliminate complications. Chart III shows a comparison, in this respect, with the three previous years and with those cases of the present season in which treatment other than sulfapyridine was used.

Empyema was present in one case, which was treated successfully by repeated aspirations. Pleural effusion was present in four of the cases and unresolved pneumonia in one.

TOXIC MANIFESTATIONS OF SULFAPYRIDINE

Reports by numerous authors give the following toxic conditions which may appear during the administration of the drug:

1. Nausea
2. Vomiting
3. Headache
4. Mental depression or confusion
5. Hiccough
6. Hepatitis
7. Acute granulocytic angina
8. Acute hemolytic anemia
9. Cyanosis
10. Oliguria
11. Hematuria
12. Calculus formation
13. Drug fever
14. Dermatitis
15. Urticaria

In the University Hospital series, the following toxic manifestations were observed:

	Number of Cases
Nausea	31
Vomiting	26
Mental confusion	3
Leukopenia	1
Severe anemia	5

In one case the nausea and vomiting were sufficiently severe to force discontinuance of the drug. Relief was obtained in four more cases of moderate severity by discontinuing the drug for one dose.

The case of leukopenia had an admission leukocyte count of 20,550 with 88 per cent polymorphonuclear leukocytes and after three days of treatment dropped to 2,400 with only 30 per cent polymorphonuclears. There was a rapid return to normal when the drug was discontinued.

In the five cases of severe anemia there was also a rapid return to normal upon withdrawal of the drug. In two cases it was judged best to give transfusions and continue the drug in view of the severity of the pneumonic process.

The following may be said concerning indications for discontinuing the use of sulfapyridine in the presence of toxic reactions to the drug. In general, nausea, vomiting, headache, mental confusion and cyanosis do not contraindicate continuance of sulfapyridine, although vomiting may render it impossible. On the other hand, a rapidly progressing leukopenia, hematuria, marked oliguria and fever resulting from the drug should indicate a cessation of its use. A moderate progressive anemia may be combatted with transfusions and the use of sulfapyridine continued if the disease warrants heroic measures. The patient should be watched very closely, however, since sudden overwhelming hemolysis with hemoglobinemia, hemoglobinuria jaundice and profound anemia may occur rapidly. Similarly, skin eruptions do not constitute an absolute contraindication but the risk of continuing the drug is certainly a serious one, especially if urticaria is present.

The contraindications to the use of sulfapyridine are usually given as follows:

1. Severe anemia.
2. A marked leukopenia.
3. Severe liver disease.
4. Severe nephritis.
5. History of previous reaction.

The relative risks to the patient of continuing or discontinuing the drug must be the criterion for decision. Cases of pneumococcus infection with anemia and hepatitis and those giving a history of previous reaction have been successfully treated with sulfapyridine, but there is no doubt that the danger of serious effects from the drug is greatly increased in these conditions.

DISCUSSION

Certainly the results with sulfapyridine in the treatment of pneumococcus pneumonia are very encouraging. It is, however, important to call attention to reports of cases of pneumococcus infection which were resistant to its action.

Various investigators suggest that in those cases in which an adequate concentration of sulfapyridine has been maintained for forty-eight hours without improvement, treatment with type specific anti-pneumococcus serum should be considered.

SUMMARY

1. Thirty-nine pneumococcus pneumonia cases in adults were treated at the University Hospital from December 28, 1938 to July 31, 1939, with no deaths. The significance of this result is analysed.

2. The effects of sulfapyridine treatment on the clinical features of the disease are reported.

3. The technic of treatment employed and the toxic reactions encountered are discussed.

The author wishes to thank Merck and Company who so generously provided the supplies of dagenan used in the early part of this investigation.

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THE TREATMENT OF PRIMARY PNEUMOCOCCIC PNEUMONIA IN INFANTS WITH SULFAPYRIDINE*†

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Pediatricians and epidemiologists have always been concerned by the high death rate occurring in infants as a result of pneumonia. Their interest has been particularly aroused when the infantile mortality rate was compared with that of the older child, in whom pneumonia results in comparatively few fatalities. The Department of Vital Statistics in 1929 estimated pneumonia as the second most frequent cause of death in individuals under five years of age, and as the most frequent cause of fatality in those of less than three years of age. Various pediatric centers (1) have reported that pneumonia accounts for a mortality rate of from 20 to 30 per cent in the infant of less than three years, whereas in the older child the mortality rate is only from 2 to 6 per cent.

A review of the pneumonias on the pediatric service of the University Hospital during a four-year period shows the striking comparison between the infant and the child in response to the disease (Chart 1). In this series the mortality rate under three years of age ranged from 20 to 27.7 per cent, while in those children from ages three to fourteen there were no fatal cases in three of the four years and an 11 per cent mortality in one year, hence an average rate of 2.75 per cent in this age bracket. The comparison is indeed striking.

Numerous therapeutic procedures have been advocated and attempted throughout the years in an effort to effect a substantial reduction in this serious disease. Despite the conscientious efforts of many workers no appreciable lessening in mortality could be effected. Transfusions and serum therapy have in the main been the most frequently advocated measures. In the opinion of the authors, which is also shared by many others, the therapeutic usage of transfusion in pneumonia is of little value except in the presence of an associated anemia. Antipneumococcic serum naturally offered a possibility of being an agent that would answer the purpose; however, the evident difficulty in typing, reactions, cost, and the absence of uniform

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response on the part of the young patient, rendered it relatively useless in the treatment of infantile pneumonia.

Recently encouraged by the results obtained by Dr. Perrin H. Long (2) in the treatment of pneumonia by the use of 2-(p-aminobenzenesulfamido) pyridine or sulfapyridine, the authors began to use it in the treatment of primary pneumococcic pneumonia.

As the literature concerning the chemical features of sulfapyridine has been so extensively presented by numerous workers, it is felt that any mention of this phase would be repetitious and a further discussion has therefore been omitted (3, 4).

METHOD OF STUDY

In attempting to determine the efficiency of any drug in the treatment of primary pneumococcic pneumonia of infants and children, the authors definitely feel that it can be evaluated satisfactorily only if it produces a marked reduction in the mortality rate of infants, as it is in this group that pneumonia constantly results in numerous deaths and is also less subject to yearly variations. Therefore, in this series the use of the drug was restricted to those infants under three years of age. Further, the reported cases consisted entirely of infants admitted to service and represented those individuals from the lower economic strata of society.

CHART 1

Mortality According to Age Groups

YEAR	BIRTH–6 MOS.			6 MOS.–12 MOS.			12 MOS.–24 MOS.			24 MOS.–36 MOS.			RATE	36 MOS.–14 YRS.		RATE
	Cases	Deaths	Rate	Cases	Deaths	Rate	Cases	Deaths	Rate	Cases	Deaths	Rate		Cases	Deaths	
			per cent			*per cent*			*per cent*			*per cent*	*per cent*			*per cent*
1934–35	2	2	100	5	0	0	6	1	16	5	2	40	27.7	40	0	0
1935–36	8	3	37	11	1	9	9	3	33	12	1	8	20	39	0	0
1936–37	3	2	66	7	2	28	8	1	12	5	0	0	21.6	26	3	11
1937–38	2	2	100	5	2	40	8	0	0	3	0	0	22.2	22	0	0
1938–39	3	0	0	13	1	8	9	0	0	9	0	0	2.9	27	0	0

Criteria for the diagnosis of pneumonia consisted of the roentgenological demonstration of a pneumonic process, no case being included in which there was not a roentgenological confirmation.

Source of material for bacterial study consisted of cultures obtained from the nares, pharynx and the blood. When pneumococci were grown in culture media, typing was attempted by means of the method employed by Vinograd, Nemir and Park (5). Blood cultures were obtained on admission and were repeated at frequent intervals while the temperature was elevated. In cases of bacteremia, cultures were taken until three consecutively negative ones were obtained.

Determinations of hemoglobin, erythrocytes, leukocytes and stained smear examinations were done daily in all treated cases.

Daily urinalysis was done, with especial attention devoted to the presence of changed or unchanged blood.

The concentration of sulfapyridine in the blood was determined daily while the drug was being used.

DOSAGE OF SULFAPYRIDINE

The amount of the drug used varied according to age. It is the authors' policy to administer an initially large dose, following which a maintenance dose is given every six hours. The initial twenty-four hour dose according to age averages approximately 1 to 1.5 grams for one year or under, 1.5 to 2 grams for one to two years, and 2 to 2.5 grams for those patients two to three years of age. The original dosage was maintained for five days after return of the temperature to normal limits, at which time the amount was halved and continued for several days. The oral route was used in all cases. The tablets were macerated in water and given to the patient in the form of a suspension. In infants the medication was placed on the posterior portion of the tongue by means of a medicine dropper. Occasionally vehicles other than water were used, viz., milk, jelly or fruit juice.

CLINICAL EFFECTS

There were thirty-four infants treated in this study, whose ages were as follows: three were less than six months of age, thirteen between six and twelve months, nine between twelve and twenty-four months, and nine between twenty-four and thirty-six months. The only fatal case occurred in the second group.

The most notable effects attributed to the sulfapyridine were shown in the temperature and clinical betterment of the patients. In all cured cases the temperature returned to normal within thirty hours. Concomitant with the reduction in fever there was noticed a marked improvement in the general appearance of the patients, restlessness was allayed, color improved and dyspnea relieved. The total leukocyte count was reduced in conjunction with the fall in temperature (6). The time consumed in reducing the leukocytes to normal was not constant; however, evidences of reduction were present within twenty-four hours. The possibility of producing an agranulocytosis was shown in case No. 6. On the fourth day of treatment the leukocytes of this patient fell to 7,600, with the polymorphonuclears giving a value of 19 per cent. The drug was immediately stopped, the patient transfused, and after seventy-two hours a normal differential count was obtained.

The blood culture in four of the cases was positive for pneumococci. Sterile cultures were obtained in all of these patients within twenty-four hours after sulfapyridine therapy was initiated.

Roentgenological examination of the chest revealed in all patients areas of consolidation, while in many evidences of a diffuse process were also present. The use of sulfapyridine did not appear to hasten resolution. This observation has also been the experience of other workers (7). The occurrence of resolution was usually several days after the institution of therapy. However, the absence of resolution did not contribute in any manner to disturbing the well-being of the patient.

Two of the cases in this study (Nos. 2 and 19) had a recurrence of pneumonia after being clinically and roentgenologically free of any pneumonic process. In case No. 2, the initial pneumonia was located in the upper right lobe. The patient exhibited a good response to sulfapyridine and roentgen examination later showed the lung fields to be clear. However, the patient remained in the hospital because of malnutrition. Twenty-two days after his initial pneumonia he had an elevation of temperature and a physical examination demonstrated a consolidated upper left lobe. Roentgen examination confirmed the physical signs. Sulfapyridine was again administered and the patient exhibited a prompt response, with amelioration of all symptoms. He was later discharged in excellent condition.

Case No. 19 had an initial upper right lobe involvement, with a pneumococcus type III being recovered. Roentgen examination on the eighth day showed the absence of any pneumonic process. Then on the tenth day, because of fever, another roentgen examination was made and a diffuse pneumonia found involving both lungs. A pneumococcus type I was recovered on this occasion. Twenty-four hours after the institution of sulfapyridine the temperature was normal and the infant made an uneventful recovery.

In this study no serious effects were encountered that could be attributed to the use of sulfapyridine other than the agranulocytic manifestation which has already been referred to and vomiting. The vomiting accompanied the immediate administration of the drug and ceased very often when the sulfapyridine was placed in admixture

CHART 2

CASE NUMBER	COLOR AND SEX	AGE	BACTERIOLOGY		DAY OF DISEASE	FEVER BEFORE THERAPY	DURATION OF FEVER AFTER THERAPY	INITIAL BLOOD LEVEL OF DRUG	COMMENT	RESULT
			Throat	Blood						
		mos.					hours	mgm.		
1	W. M.	4	Pneumococcus Type 22	0	3rd	103°	24	6.4	Acute Otitis Media on admission. Very toxic, respirations were shallow.	Recovered
2	B. M.	4	Pneumococcus	0	3rd	102°	24	13.8	Pneumonia on admission in right upper lobe. 22 days after recovery, second pneumonia in both upper lobes.	Recovered
			Pneumococcus	0	2nd	103°	8	11.1		
3	B. M.	5	Pneumococcus	0	2nd	102.6°	30	10.5	No complications.	Recovered
4	B. M.	7	Pneumococcus Type 6	0	2nd	104°	30	1.5	Left myringotomy for Suppurative Otitis Media on admission. Marked toxicity.	Recovered
5	Y. F.	7	Pneumococcus Staph. albus hem.	0	5th	104°	18	7.5	No complications.	Recovered
6	W. F.	7	Pneumococcus	0	2nd	101°	24	9.4	Drug stopped on 4th day because of W.B.C. = 7600, P.M.N. = 19%.	Recovered
7	W. M.	8	Pneumococcus Strept. hem.	Pneumococcus	2nd	105.4°	24	4.1	Acute Otitis Media on admission. Blood culture was negative on 2nd day of therapy.	Recovered
8	W. M.	8	Pneumococcus	0	4th	101.6°	24	12.3	Right myringotomy for Suppurative Otitis Media on admission.	Recovered
			Type 11						Extremely toxic, moderately cyanotic, dyspneic.	
			Strept. hem.						Recovery rapid.	
9	W. M.	8	Pneumococcus Staph. albus hem.	0	3rd	103.8°	120	10.8	Marked toxicity, cyanosis, moderate dyspnea. Clinically improved rapidly. Sudden death on 7th day. Autopsied—see text.	Died
10	W. M.	8	Pneumococcus	0	3rd	105°	30	3.3	Very toxic, respirations labored. Acute Otitis Media on 5th day. Myringotomy performed.	Recovered
11	B. F.	8	Pneumococcus	0	4th	103.8°	18	9.3	Left draining Otitis Media on admission.	Recovered
12	W. F.	9	Pneumococcus	0	3rd	102.4°	18	2.4	Acute Otitis Media on admission.	Recovered
13	B. F.	12	Pneumococcus	0	3rd	102.4°	24	6.8	Right myringotomy for Suppurative Otitis Media on admission.	Recovered
14	B. M.	12	Pneumococcus	0	?	104°	24	10.5	Right draining Otitis Media on admission.	Recovered
15	B. M.	12	Pneumococcus	0	?	103°	24	4.4	No complications.	Recovered

16	B. F.	12	Pneumococcus	0	4th	102.6°	36	4.9	Moderately labored respirations. Acute Otitis Media on admission. Myringotomy on right on 3rd day.	Recovered
17	W. F.	13	Pneumococcus	0	3rd	104.2°	12	1.2	Moderate toxicity.	Recovered
18	W. M.	14	Pneumococcus Type 9	0	5th	104.2°	30	5.0	Marked toxicity.	Recovered
19	W. M.	14	Pneumococcus	0	4th	101.2°	24	4.4	Acute Otitis Media on admission. Pneumonia in right upper lobe.	Recovered
			Type 3						On 8th day, x-ray of chest showed same to be clear.	
			Type 1	0	1st	102.4°	24	5.1	On 10th day, second pneumonia began in left lower lobe.	Recovered
20	W. M.	15	Pneumococcus	0	3rd	104.4°	24	6.1	Acute Otitis Media on admission. Marked toxicity, slight cyanosis.	Recovered
			Type 14						On 5th day patient had left-sided convulsion, high fever, relieved by bilateral myringotomy. Lumbar puncture was negative.	
21	B. M.	15	Pneumococcus	0	4th	103.8°	24	4.8	Moderate cyanosis, toxicity. Acute Otitis Media on admission.	Recovered
22	W. M.	17	Pneumococcus Type 14	0	5th	102.8°	24	6.5	Bilateral draining Otitis Media on admission	Recovered
23	W. F.	18	Pneumococcus Type 4	0	3rd	104.8°	24	10.0	Left myringotomy for Suppurative Otitis Media on admission.	Recovered
24	W. M.	19	Pneumococcus	0	3rd	104.8°	18	5.1	Acute Otitis Media on admission.	Recovered
25	B. F.	21	Pneumococcus	0	5th	105.2°	24	7.8	Marked toxicity.	Recovered
26	W. M.	24	Pneumococcus	Pneumococcus	2nd	103°	24	4.6	Extremely toxic. Blood sterile on 2nd day of therapy.	Recovered
27	B. F.	24	Pneumococcus	0	2nd	104.8°	18	2.3	Meningismus associated with marked toxicity.	Recovered
28	W. M.	30	Pneumococcus	0	2nd	102.4°	30	2.2	Acute Otitis Media on admission.	Recovered
29	B. F.	30	Pneumococcus	0	3rd	103°	24	6.8	Moderate toxicity. Acute Otitis Media on admission.	Recovered
30	W. F.	30	Pneumococcus	Pneumococcus	3rd	103.4°	24	5.5	Marked toxicity. Blood was sterile on 2nd of therapy.	Recovered
			Type 1	Type 1					See text for subsequent hospital course.	
31	B. M.	36	Pneumococcus	0	5th	105°	24	6.1	Acute Otitis Media on admission.	Recovered
32	B. F.	36	Pneumococcus	0	4th	102.4°	12	6.9	Acute Otitis Media on admission.	Recovered
33	W. M.	36	Pneumococcus	0	3rd	106°	30	4.6	Irrational. Otitis Media on admission. Myringotomy on 10th day.	Recovered
34	B. F.	36	Pneumococcus Type 1	Pneumococcus Type 1	4th	101.8°	18	2.2	Blood culture was sterile on 2nd day of therapy.	Recovered

with jelly or some such vehicle. In no case did the vomiting assume any degree of severity.

The blood concentration of sulfapyridine was determined daily in all cases while under treatment. The range was from 1.2 mg. per cent to 13.8 mg. per cent. Hence, with such a wide range of concentration no conclusion can be reached as to the optimal concentration. The patient in Case No. 17, in whom the blood level was 1.2 mg. per cent, had a return to normal temperature twelve hours after therapy was instituted and that was only the third day of the pneumonia.

COMPLICATIONS

During this study there occurred only three cases of otitis media while the drug was being given or shortly after it had been discontinued. In case No. 10 otitis developed on the fifth hospital day, myringotomy was performed and recovery was rapid and uncomplicated. Case No. 20 entered with right suppurative otitis media. On the fifth hospital day the patient had an elevation of fever and shortly thereafter a left-sided convulsion lasting only ten minutes. Physical examination revealed only an acute left otitis media. The eye grounds were normal to ophthalmoscopic examination. Lumbar puncture yielded spinal fluid under normal pressure and no increase in cells. Myringotomy was performed and the temperature returned to normal limits within twelve hours. In case No. 33 a right myringotomy was performed on the tenth hospital day, with an uneventful recovery.

There were no cases of empyema encountered in this series. The notorious yearly variation of empyema makes it imperative that no conclusions be drawn concerning the effect of sulfapyridine on the incidence of empyema.

THE FATAL CASE

The only fatality resulting from pneumonia in this series occurred in case No. 9. This eight-month old infant had a hemolytic staphylococcus albus in addition to a pneumococcus (type undetermined) recovered on pharyngeal culture. The temperature in the patient returned to normal limits two days before death. At post mortem examination very little was found other than the findings of a resolving pneumonia, atelectasis, thick mucoid bronchial secretions and cloudy swelling of all viscera. There were no findings in which sulfapyridine could be indicated as having been a causative factor in the death of this patient.

DISCUSSION

Since the introduction of sulfapyridine, numerous publications have appeared concerning the effect of sulfapyridine on the pneumonia of childhood. All the results have been in agreement that it is an agent which is highly effective in the treatment of pneumococcal pneumonia and that it has been responsible in reducing the mortality resulting from this disease to a level never before attained.

Unfortunately, the drug has been administered by previous workers to children regardless of age. Failure to place any limitation on age naturally gives rise to the criticism that the results of these workers could have been caused by coincidence rather than by any specific action of the drug. One readily appreciates a criticism of this nature when consideration is given to the extreme variability of mortality resulting from pneumonia in the older child, as has been expressed in the introductory paragraph of this article. While the element of chance in the authors' opinion seemed of a necessity slight, it is felt that they have lessened the force of this criticism by restricting the use of sulfapyridine to an age group in whom pneumonia has always been an extremely serious disease. This statement is better appreciated when one considers that the authors had only one fatality in a series of thirty-four cases, which when reduced results in a total mortality percentage of 2.9 per cent, a figure far below any previous year encountered in this restricted age group.

It is very pleasing to note the absence of any fatalities in the age group from birth to six months. While the number of cases were few during this period, in previous years the authors had an equally small number of cases but were not so fortunate as to have all of these infants recover.

Criticism might be directed as to the institution of sulfapyridine therapy in regard to the day of the pneumonia. The average day of beginning treatment was 3.2 days. The range was from eight hours to five days, with the majority starting treatment on the third day. These results suggest that sulfapyridine therapy alone was responsible for the rapid recovery, as the occurrence of an average of 4.3 days for a pneumococcic pneumonia in a series of infants hithertofore would have been rare indeed.

The authors necessarily recognize the limited value of arriving at conclusions in any therapeutic procedure attempted in pneumonia when based on a relatively small series of cases. The necessity of further study as to possible undesirable or unrecognized reactions is likewise admitted.

It seems quite logical at present to consider sulfapyridine as an extremely useful weapon to add to the antipneumococcal armamentarium, especially when one has effected a reduction in the mortality from primary pneumococcic pneumonia of infants so far below any previous yearly figure that to consider it the result of coincidence rather than the actual effect of the drug would seemingly be an untenable view.

Since the completion of the above report, there have been admitted and successfully treated with sulfapyridine twenty-five additional cases of primary pneumococcic pneumonia in the same age group, with no mortalities.

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EXPERIMENTAL OIL EMBOLISM*†

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Much has been written concerning fat and oil embolisms since 1669, when Lower injected milk intravenously into dogs. Following this, there was a long hiatus in the work until Magendie injected olive oil into the veins of animals and saw the mechanical occlusion of the smaller lung capillaries, which had produced rales, fever and dyspnea. In 1862, Virchow repeated Magendie's experiments and confirmed them. Müller (1860) was the first to describe fat embolism in man. He saw globules of fat in the choroidal capillaries of a young man dying of chronic nephritis. In the same year, Cohn noted fat emboli in the capillaries of the brain. Zenker (1862) found fat embolism in the lungs of a man, following a severe crushing injury to the chest. Von Bergmann (1863), by injecting warm lard into the saphenous veins of cats, observed obstruction of the pulmonary capillaries with rupture and the subsequent liberation of fat into the alveoli and dilatation of the right side of the heart. Between 1863 and 1873 many papers appeared on this subject, the most important of which was that by Busch, who found that upon traumatizing bone, fat emboli could be found in the lungs. He also discovered, by the use of cinnabar, that absorption of the fat in the lungs was rapid. This rapid absorption was later confirmed by other workers (Fritzsche, Caldwell and Huber). Von Bergmann first diagnosed fat embolism in man. In 1877, Riedel stated that all bone injuries led to fat embolism of variable degrees.

Scriba (1880) was the first to publish a comprehensive paper on fat embolism, and summarized the literature together with his own and other experimental work.

Hamig called attention to the latent period between the respiratory symptoms and cerebral manifestations.

Warthin's article completed an exhaustive study of the situation up to the date of its publication. He not only reviewed the history, but also the pathology, symptomatology and treatment. He described the lesions in the lungs, brain and kidneys and called attention to two groups of symptoms: respiratory, characterized by dyspnea, cough,

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cyanosis, pulmonary edema (with moist rales over the bases); cerebral, conspicuous by a latent interval of three to eight days, uneasiness, headache, stupor, tremors, convulsions and high fever.

Since this outstanding article over 150 contributions have appeared, but almost all of them have been limited to case reports or facts already known. However, certain articles did appear which pushed the frontiers forward.

Wegelin and others showed that lymphatic transportation of fat was negligible.

Paul and Windholz found that they were able to produce a tolerance by giving small doses intravenously. At intervals, the animals so treated could stand many times the dose necessary to kill untreated animals. This was not confirmed by Lehman and Moore. These latter investigators also pointed out that ultramicroscopic fat particles in emulsion in the blood plasma could be coarsened. This coarsening was produced in vitro by fat solvents and soap precipitants of protein decomposition, and in vivo by the use of ether intravenously or its prolonged inhalation. Besides, they stated, there was a constant relationship between the amount of fat found at necropsy and the severity of the symptoms.

Beneke suggested that fat is broken up in variously sized particles not by mechanical means alone, but also by saponification and emulsification.

Holt stated that emulsions specially prepared with lecithin, in which the globules of fat were two microns in size, had little effect in children when injected intravenously.

Susani, after studying the relationship between the size of the oil droplets and the lethal dose in animals, concluded that the lethal dose, as determined by the actual amount of oil required, was less when the oil was emulsified into droplets measuring 100 to 200 microns than it was when pure oil was used. He also thought in the matter of droplets of less than 100 microns in diameter, the finer the emulsion the greater the total amount of oil required to kill.

The purposes of the following experiments are: 1. To determine the effect of the degree of dispersion of oil upon the minimal lethal dose. 2. To determine whether the oil droplets, when injected into the circulation, undergo any change in size.

METHODS

In these experiments, mineral oil was used as the embolic agent. This oil was selected because it is readily emulsified and forms a fairly stable emulsion. Moreover, mineral oil is readily differentiated from animal fat and since it is not absorbed, the emboli remain in situ for a considerable time following their lodgement.

TABLE 1

Pure Mineral Oil

RABBIT	AMOUNT OF OIL	RESULTS
#1	0.8 cc./kg.	Died in 4 minutes
#2	0.6 cc./kg.	Found dead next morning
#3	0.6 cc./kg.	Died in 6 minutes
#4	0.5 cc./kg.	Killed at end of 15 days
#5	0.5 cc./kg.	“ “ “ “ 12 “
#6	0.4 cc./kg.	“ “ “ “ 12 “
#7	0.2 cc./kg.	“ “ “ “ 12 “
#8	0.1 cc./kg.	“ “ “ “ 13 “

TABLE 2

Tragacanth Emulsion—Globules 50–80 Microns

(Amounts expressed in terms of pure oil)

RABBIT	AMOUNT OF OIL	RESULTS
#22	0.4 cc./kg.	Died at end of 12 hours
#23	0.4 cc./kg.	“ during injection
#24	0.3 cc./kg.	“ at end of 1 hr., 20 min.
#25	0.3 cc./kg.	“ “ “ “ 25 minutes
#26	0.3 cc./kg.	“ “ “ “ 3 days
#27	0.3 cc./kg.	“ “ “ “ 5 hours
#28	0.2 cc./kg.	Killed at end of 15 days
#29	0.1 cc./kg.	“ “ “ “ 15 “

TABLE 3

Acacia Emulsion—Globules 31–43 Microns

(Amounts expressed in terms of pure oil)

RABBIT	AMOUNT OF OIL	RESULTS
#30	0.6 cc./kg.	Died at end of 6 minutes
#31	0.5 cc./kg.	“ “ “ “ 10 “
#32	0.4 cc./kg.	“ “ “ “ 40 “
#33	0.4 cc./kg.	“ “ “ “ 4 “
#34	0.3 cc./kg.	“ “ “ “ 12 days
#35	0.3 cc./kg.	“ “ “ “ 3 hours
#36	0.2 cc./kg.	Killed at end of 13 days
#37	0.2 cc./kg.	“ “ “ “ 15 “

In the preparation of the emulsions, acacia and tragacanth were used as emulsifying agents. The primary acacia emulsion was prepared by the continental method, using acacia 6.2 gms., water 12.5 cc. and mineral oil 25 cc. The primary emulsion was diluted with water to 100 cc. The tragacanth emulsion was prepared in a similar manner, with .65 gms. of tragacanth as the emulsifying agent.

The homogenized emulsion was prepared in a homogenizer, and acacia used as the emulsifying agent.

The size of the oil globules in the various emulsions was determined by measurement with an eyepiece micrometer. The vast majority of globules in the acacia emulsion varied between 31 to 43 microns in diameter; in each microscopic field, however, there were a number of smaller globules. In the tragacanth emulsion, the majority of the globules varied from 50 to 80 microns, but here, too, smaller globules were present. The homogenized emulsion contained the most uniform globules, the average diameter of which was 6 to 9 microns.

TABLE 4

Homogenized Emulsion—Globules 6–9 Microns

(Amounts expressed in terms of pure oil)

RABBIT	AMOUNT OF OIL	RESULTS
#9	0.8 cc./kg.	Died at end of 24 hours
#10	0.8 cc./kg.	" " " " 50 minutes
#11	0.7 cc./kg.	" " " " 3 hours
#12	0.6 cc./kg.	" " " " 6 minutes
#13	0.6 cc./kg.	" " " " 2 hours
#14	0.5 cc./kg.	" " " " 2 hrs., 21 min.
#15	0.5 cc./kg.	Killed at end of 14 days
#16	0.5 cc./kg.	Died at end of 2 hrs., 17 min.
#17	0.4 cc./kg.	Killed at end of 17 days
#18	0.4 cc./kg.	" " " " 14 "
#19	0.3 cc./kg.	" " " " 12 "
#20	0.2 cc./kg.	" " " " 17 "
#21	0.1 cc./kg.	" " " " 17 "

In these experiments, rabbits were used exclusively. The injections of oil and oil emulsions were made into the marginal ear veins.

RESULTS

The table on the following page shows in a comparative way the minimal lethal doses for pure oil and the various emulsions.

Autopsies were performed on all animals. Frozen and paraffin sections were studied in each case. For the identification and study of the emboli, frozen sections were stained with scarlet R.

In animals dying during or shortly after injection, embolism was confined almost entirely to the vessels of the lungs. Where pure oil was injected it was obvious from sections that, while there was dispersion, the bulk of the oil remained in some of the larger pulmonary vessels. With the homogenized oil fine globules were found in small capillaries in the lung and as compared to the pure oil, acacia and tragacanth emulsions, the emboli were more widely distributed throughout the general circulation. Measurements of the emboli showed that there had been little change in the size of the oil droplets injected into the circulation.

In an attempt to study the disposition of oil droplets in the circulation of rabbits, emulsions of lipiodol and pure lipiodol were injected. Opacities in x-ray films were seen only in animals receiving the pure oil.

TABLE 5

EQUIVALENT IN PURE OIL	PURE OIL	HOMOGENIZED EMULSION	ACACIA EMULSION	TRAGACANTH EMULSION
0.8 cc./kg.	D	D D		
0.7 cc./kg.		D		
0.6 cc./kg.	D D	D D	D	
0.5 cc./kg.	L L	D L D	D	
0.4 cc./kg.	L	L L	D D	D D
0.3 cc./kg.		L	D L	D D D D
0.2 cc./kg.	L	L	L	L L
0.1 cc./kg.	L	L	L	L

D—Animals dying as result of injection.
L—Animals surviving.

DISCUSSION AND SUMMARY

By the use of mineral oil as an embolic agent in a relatively small series of rabbits it has been shown that the approximate minimal lethal dose for pure oil is 0.6 cc. per kg. of animal weight; for homogenized oil the minimal lethal dose lies between 0.5 cc. and 0.6 cc. per kg.; for the acacia emulsion between 0.3 cc. and 0.4 cc. per kg., and for the tragacanth emulsion 0.3 cc. per kg. The minimal lethal doses of pure oil and of homogenized oil are approximately the same and they are larger than the minimal lethal doses of the acacia and tragacanth emulsions. From the study of the sections it appears that the pure oil tends to collect in larger pulmonary vessels. The homogenized oil, because of the small size of the droplets, 6 to 9 microns, passes readily into the wide capillary circulation of the lung and the droplets find much less difficulty in entering the greater circulation. The droplets of the acacia and tragacanth emulsions, measuring from

31 to 80 microns, because of their size do not pass readily through the capillary circulation of the lung and hence produce greater obstruction than do either pure oil or the homogenized emulsion. It would appear that globules that range between 31 to 80 microns all have about the same effect in the matter of the amount of oil necessary to produce death in animals. The globules of mineral oil tend to retain their original size within the circulation of rabbits and there is no evidence to indicate that the oil globules are added to from the fat of the blood.

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POLYCYSTIC DISEASE*†

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INTRODUCTION

This paper is designed to bring to the reader the pertinent facts concerning polycystic disease, more especially that of the kidney and liver, a review of the recent literature, and a series of cases found in the University and Johns Hopkins Hospitals, with special regard to symptoms and diagnosis.

DEFINITION

Polycystic disease is a term applied to the congenital condition which is characterized by the formation of numerous cysts in the kidneys, sometimes in the liver, and rarely in the pancreas and the spleen.

ETIOLOGY

Ribbert's theory states that the cyst is caused by interference with the union of the glomerular part of the tubule with the other rudiment which ascends from the ureter to join it, hence the glomerular portion dilates into a cyst. The end of the ureteral portion also may become cystic. Others regard the whole process as an adenomatous growth which may account for a similar growth in the liver.

Kampmeier (1) believes that cyst formation is the result of an incomplete involution of the first, second, third and fourth generations of convoluted tubules. In other words, he appears to have established an embryological sequence consisting of a theory of generations of uniferous tubules before the final normal product is elaborated. The earlier discarded generations become vestigial but may persist for a long time, and, as a rule, pass through a stage characterized by a cystic formation. At the end of their allotted time these rudimentary cystic tubules become suppressed and disappear.

In some instances the unknown factor which controls the normal degeneration of such rudimentary structures may fail to operate or fail to operate sufficiently. A single vestigial tubule may grow, or if there is a total failure of arrest all cystic tubules will persist and expand and the polycystic kidney will eventuate.

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Teuscher has suggested that a cystic formation of the liver depends upon an anomaly in the germ plasm. In examining the liver, she always found openings between the cysts and bile passages and concluded, therefore, that the cyst formation was not caused by the failure of the ducts to establish these connections with the larger bile passages, but by a capacity for growth on the part of the epithelium. She suggested that the cystic development was an expression of the perverted tendency, widespread in the body, of the tissues of the excretory ducts to multiply at the expense of the more highly differentiated glandular tissue.

Carling and Hick have noted the histological resemblance between cysts of the liver and kidney and cystadenomas of the pancreas. They consider that the association of the polycystic disease of the liver, pancreas and kidney can be explained by assuming the inclusion of isolated portions of the Wolffian body either in the enteric bud forming the liver and pancreas as it develops in the ventral mesogastrium, or of that portion of the intermediate cell mass in which the kidney forms. It seems reasonable to suppose that the cases seen in adult life are the milder forms of the condition, and since it is a malformation the process is not necessarily progressive.

Thus one can see there is marked diversity of opinion and no one theory is generally accepted.

INCIDENCE

Tallam states that polycystic degeneration is rare, being observed only eighteen times in the past twenty-six years at the Federoff Clinic. Statistics gathered from other institutions showed only sixteen cases in 10,177 autopsies. Bell reports that this disease is recognized clinically once in every 3,523 hospital admissions, and that lesions are found in one out of every 500 autopsies.

At the University Hospital the results were somewhat different. There were seventeen cases out of 99,054 admissions, or one to every 5,824 admissions. According to a recent article by Spencer (5) of the same institution, there is an incidence of one case in every 228 autopsies. In the series taken from this clinic, only two out of seventeen cases reported showed cysts of the liver as proved by autopsy findings. In addition, there were two other cases of congenital cystic liver without any renal involvement found at the time of routine autopsy.

At the Johns Hopkins Hospital eight cases were found indexed under polycystic kidney. However, this cannot be accepted as a complete record as there are one or more departments, mainly the department of gynecology, which have not been indexed for ten years.

There have been several methods of indexing employed, therefore the present system is not complete. Three of these cases were autopsied. Two were found to have polycystic livers and kidneys, and in addition one of these two cases had a polycystic pancreas. The third case showed only kidney involvement.

Incidence of polycystic liver. In adults polycystic disease of the liver is frequently but not invariably associated with cystic disease of the kidneys, and in some cases there have also been present cysts in the pancreas and spleen. Clinically, according to Rollingstone, the cases may be divided into those found in infants and those found in adults. In infants the majority of cases reported by Rollingstone were associated with other deformities, the most constant of which were cystic or imperfectly developed kidneys and polydactylism.

It is suggested that the condition is probably overlooked and if sought for in monsters and stillborn infants it might be found more frequently than the number of recorded cases indicate.

Sex. In the seventeen cases at the University Hospital, eight males and nine females were afflicted, while the Johns Hopkins Hospital had four cases of each sex.

Race. There were six colored and eleven white cases at the University Hospital, while all cases at the Johns Hopkins Hospital were white. According to this report the distribution among sexes is about equal, which is in accordance with the reports of many other clinics. On first consideration there is apparently a greater number of these cases in white people, but when one considers the ratio of white to colored admissions in a general hospital, the incidence is probably about equal.

Age. An analysis of these cases according to age incidence shows that the majority present themselves for treatment between the ages of thirty and fifty. However, notice must be taken that three of the twenty-five cases at both hospitals occurred below one year of age and that symptoms may be noted at any age.

Heredity. Heredity plays an important role in congenital polycystic disease. Dunger observed the disease in five siblings. Cairns (2) found ten cases in three generations of the same family. Shapiro (3) found the disease in a mother, four of her children and one of her grandchildren. Goldstein (4) reported the disease in a mother and four of her children, and in a father and two of his children. Teuscher reported a case in a family where two members are known to have suffered from polycystic disease and five others were strongly suspected.

PATHOLOGY

Spencer (5) states that the gross pathology of the kidneys is striking. The organs are usually enlarged and although weights up to 3,000 grams and more have been recorded, the weight of the average polycystic kidney is 500 grams. There is frequently some difference in the size of the affected kidney. In a particular individual and in 5 to 10 per cent of the cases the lesion is unilateral. The kidney in spite of marked enlargement retains a relatively normal shape. The exterior is studded with round cysts. On gross sections the cut surface presents a honeycombed appearance and the renal parenchyma is largely replaced by cysts, which vary in size from 2 to 3 mm. up to 3 to 4 cm. or more in diameter. The walls of the cysts are relatively thin and the cysts contain yellow gelatinous material or fluid which may be clear, dark brown or turbid. The fluid in the cyst is rich in urea and uric acid, and small grayish islands of renal parenchyma may be observed in the intercystic spaces. In the advanced cases these islands are most abundant in the subcapsular zone. The pelvis and ureters are pushed down and toward the midline. The pelvis is also elongated.

Histologically the small cysts are lined with small clear cuboidal epithelial cells, while in the larger cysts because of pressure the epithelium is often markedly flattened. Occasionally glomeruli may be seen projecting into the cyst, the remaining renal parenchyma is compressed, and throughout the kidney there is scarring. The arteries commonly show intimal thickening, and in advanced cases with hypertension the arterioles are involved.

In polycystic disease of the liver, this organ is usually enlarged and presents a varying number of bluish-gray, grapelike cystic masses. They may vary in number from a very few localized cysts to many areas where the liver is honeycombed with these cysts. The largest cysts at times may appear inflamed and may adhere to the parietal peritoneum. The cysts may vary in size from microscopic to quite large. Marrin in the Irish Journal of Medical Science reports a case in which there were many cysts as large as tennis balls.

There is, as a rule, no line of cleavage between the cystic wall and the liver substance which would permit enucleation. The fluid in the cyst is usually clear, of a faint straw color, and contains no bile. It is rich in albumin and contains some urea, cholesterin and sodium chloride. The fluid generally shows some red blood cells and leukocytes. Upon microscopic examination a section of such a liver contains an abundance of fibrous tissue. The essential liver cells appear as a scattered group of cells in a stroma of fibrous tissue. The lining cells of the cysts are of cubical epithelium in the smaller cysts but appear flattened in larger cysts, probably because of pressure. Normal bile ducts are seen beside the cyst.

SYMPTOMS AND SIGNS

The more important symptoms are pain in the flank or epigastrium, enlargement of the kidney, hematuria, and signs of chronic nephritis with accompanying uremia.

1. Pain is by far the most frequent early symptom. It marked the onset in seventeen of the twenty-five cases reviewed in this paper. The pain which is caused by the enlargement is usually felt in the region of the kidney. It is dragging in character and often increased by exertion or relieved by lying down. At first it is paroxysmal and later continuous. When hematuria is present the passage of a clot may produce pain which is characteristic of a renal colic. Occasionally the location of the pain is atypical and occurs in the lower chest and axilla.

2. Kidney enlargement is another of the most common symptoms which cause the patient to come to the physician; the enlargement is usually progressive and in thirteen of the twenty-five cases in this series it was an outstanding complaint and symptom. However, contrary to the accepted belief, the tumor mass is palpable on one side only in many cases. The enlarged kidney is frequently tender to palpation, especially in cases where there is infection present. Enlargement tends to push the colon forward or to the inner side. In these cases resonance may be determined over the tumor mass. In thin patients the irregular surface of the projecting cyst can be palpated. These enlargements also produce one of the most frequent complications, a ptosis producing a hydronephrosis by angulation of the ureter.

3. Hematuria occurs at one time or another in most cases and may be the initial symptom. It tends to be intermittent, is often profuse and sometimes fatal. Most workers have found this symptom in 33 per cent of their cases. The authors have found it in 64 per cent of their cases; however, these were both microscopic and macroscopic hematuria.

4. Albuminuria is found consistently, while casts were found in only 36 per cent of the cases.

5. Hypertension, although not an early symptom, was found to be present in more than 50 per cent of the cases in this series at the time of clinical diagnosis. The basis used was a blood pressure of 160/90 or higher. Later in the disease, after renal failure, it is evident that

hypertension is a constant finding, which would indicate that this symptom is for the most part secondary to kidney damage. Such symptoms as headache, cerebral accident, cardiac enlargement, edema, dyspnea, and so forth, are all secondary to the hypertension and are present in a number of the cases.

6. Anemia: Secondary anemia in varying degrees was present in 50 per cent of the cases, especially in those where kidney failure was evident.

7. Polyuria and frequency are here, as in any other chronic nephritis, evidence of early kidney damage. Specific gravity of the urine is usually fixed and at a low level. Phthalein and urea clearance tests are always impaired. Phthalein in two of the cases was of too small a value to read.

8. Uremia: When the blood nonprotein nitrogen begins to rise as a result of renal failure, one realizes that the functional tissue of the kidney has been replaced to such a degree by numerous cysts that uremia is inevitable. This was shown in 40 per cent of the cases reviewed. An interesting but striking feature of polycystic disease is the tolerance these patients have for a high nonprotein nitrogen retention without showing any symptoms. Uremia is the most frequent cause of exit, thus its appearance is of grave prognostic importance.

SYMPTOMS OF POLYCYSTIC LIVER

Clinically polycystic liver gives no definite symptoms. Where there is associated disease of the kidney, the condition might be suspected if the symptoms of uremia were associated with an enlarged and nodular liver. A single large cyst may simulate any abdominal tumor. As a rule, in polycystic disease of the liver there is no jaundice, no symptoms of biliary obstruction and no digestive disturbances are present, and judging by functional tests the pigmentary glycogenic and excretory functions of the liver remain unaffected.

It is of interest to report that in one case of this series a patient with polycystic kidney and polycystic liver developed ascites as a result of the pressure on the portal system by the multitude of cysts of the liver. This is an exception rather than the rule and if it were not for the associated kidney damage, most of these cases of polycystic liver would remain undiagnosed because of the lack of symptoms. As evidence of proof two cases of polycystic disease of the liver alone were noted which remained undiagnosed until the time of operation and autopsy.

UROGRAPHY

Of all the technical procedures, urography as done by the retrograde method is the greatest single diagnostic procedure. Needless to say, intravenous pyelography is valueless because of the impaired kidney function.

According to Spencer (5), the characteristic roentgenological findings are: low position of the kidney; a longitudinal stretching of primary calices, especially the highest and the lowest; elongation of the pelvis; blunting of the minor calices; right angulation of the ureter in relation to the pelvis; displacement of the ureter to the midline, and bilateral pelvic changes.

A good precaution to observe when polycystic disease of the kidney is suspected is to do only one pyelogram at the time of the first cystoscopic examination, as patients with borderline kidney function may be thrown into uremia by a bilateral pyelogram.

CONDITIONS TO BE CONSIDERED IN THE DIFFERENTIAL DIAGNOSIS OF POLYCYSTIC DISEASE OF THE KIDNEY

The conditions to be considered in differential diagnosis of the polycystic kidney are: solitary cysts; chronic nephritis which frequently produces numerous small cortical cysts; dermoid cysts; echinococcus cysts; paranephritis cysts; kidney tumors.

The clinical manifestations are usually not seen until 40 to 50 years of age, although they are present from birth. Commonly the process is more advanced in one kidney than the other and even though one organ may be normal in gross appearance it will be found to be basically cystic, and this dormant tendency will rapidly assume activity if imprudent radical surgery is undertaken on the other side. Not infrequently great enlargement takes place and the abdomen may become filled with bilateral knobbed masses. This advanced stage may present itself quite early in life in some instances. As long as sufficient functioning tissue persists, and gross masses are not obtrusive or complications do not arise, there may be few if any symptoms and the patient may enjoy long life without suspecting his real condition. More generally the picture of progressive renal insufficiency supervenes and the diagnosis of chronic nephritis is made.

There may be headache, lassitude, digestive disturbances, anemia, some elevation of blood pressure, changes in ocular fundi, dull pain in the back and spontaneous varicocele. Hypertension or cardiac failure when present will introduce other symptoms including cerebral hemorrhage, dyspnea and edema. The case may proceed insidiously until the point of almost total renal failure is reached and suddenly the patient passes into a state of terminal uremia.

The laboratory data depend on various factors, especially urinalysis, which may be almost negative or may show pus and blood in small or large quantities. Casts occur but are not as common as would be expected. Low specific gravity is a frequent finding. The blood chemistry may remain normal for a long time and the more sensitive dye test or the urea clearance test is coincidentally indicative of marked depression of function.

In later stages, the blood chemistry is significantly altered. The urographic data are great and usually of determining importance as far as the diagnosis is concerned, although it may closely simulate the type produced by neoplasm. The pyelogram of the polycystic kidney is fairly characteristic and is conclusive when investigation discloses similar findings on the opposite side. It is well to point out again that bilateral simultaneous pyelography may precipitate uremia and should never be done. Complications are prone to occur, especially from hemorrhage, acute or chronic infection, or from severe pain caused by tension within the kidney or pressure on adjacent structures. The clinical picture is altered accordingly.

The solitary cystic kidney carries with it no such significant train of events. It is usually unilateral and progressive. Deterioration of renal activity, with its multitude of sequelae does not occur in this condition because there is a large residue of active functioning kidney tissue. Solitary cystic kidneys are found in 3 to 5 per cent of all autopsies and are quite uncommon clinically. Young reported not a solitary cyst in 12,500 cases. Stevens of San Francisco County Hospital recorded only two cases in 58,288 consecutive admissions. In 1932 McKay could find only 256 cases in the entire medical literature and by far the greater number were found by pathologists and not by surgeons.

The urinalysis is generally negative in the absence of complications such as hemorrhage or infection. Urography will be of no aid unless the cyst impinges upon the pelvis, in which event a characteristic deformity will exist. A flat plate often reveals a cystic mass in the renal areas. Echinococcus cysts, dermoid cysts and paranephritic cysts can be dismissed with the statement that they are rare, unilateral, and do not produce the above characteristic picture. Chronic nephritis should not cause confusion owing to the absence of palpable masses, coexisting polycystic disease of the liver, and the absence of the characteristic pyelogram. Neoplasms of the kidney are usually unilateral and do not show the characteristic bilateral pyelography.

CONDITIONS TO BE CONSIDERED IN THE DIFFERENTIAL DIAGNOSIS OF POLYCYSTIC DISEASE OF THE LIVER

The conditions to be considered in the differential diagnosis of polycystic disease of the liver are: ciliated epithelial cysts; teratomatous cysts; pseudocysts; cysts arising from blood of the lymphatic vessel; echinococcus cysts; simple cysts. Ciliated epithelial cysts, teratomatous cysts, pseudocysts and cysts arising from blood or lymphatic vessels are so rare that they will not be considered. Echinococcus cysts can be differentiated from polycystic disease of the liver only by serological test, unless fluid from one of the cysts is obtained. Simple cysts are usually unilocular and are unassociated with polycystic disease of the kidney.

COMPLICATIONS

The following are the more important and prominent complications of polycystic disease of the kidney: hemorrhage; suppuration; hydronephrosis; pyelonephrosis; rupture of a cyst; perforation of a suppurative cyst followed by perirenal cellulitis; acute anuria.

The complications of polycystic disease of the liver are extremely rare but those that have been recorded are as follows: hemorrhage into a cyst; suppuration; rupture; strangulation of a pedicled cyst.

PROGNOSIS

Prognosis in cases of polycystic disease without symptoms is indefinite. There are doubtless a number of cases which have a normal span of life with this affliction. On the other hand, many die in the early months of life from obscure causes and if autopsied they would be found suffering from polycystic disease. The duration of life from the onset of symptoms may be from one year to, in exceptional cases, as long as twenty years.

TREATMENT

The treatment of polycystic disease of the kidney is fundamentally conservative by force of circumstances. The bilaterality of polycystic disease prohibits radical surgery or surgery of any description if it can be possibly avoided. Complications may appear to invite intervention but usually this is undesirable. Throughout the literature is given the warning against interference in these cases. However, more recently there has been a trend in the opposite direction. In the opinion of Meltzer and Brasch, lesser indications such as pain and hemorrhage may be sufficient indication for operation, nephropexy, puncture, or enucleation of one or more cysts. The value of such procedure is sharply protested in other quarters and the weight of opinion is decidedly on the side of inaction unless a more dramatic situation arises and actually threatens the life of the patient. These more serious complications are the development of tuberculosis (6), a malignant tumor, or a calculous pyonephrosis in the congenital cystic kidney. Therefore, these types of cases are indications for urgent nephrectomy. The mortality in these circumstances is high, usually about 30 per cent. The majority of patients die in a few months or years after operation. Certainly nephrectomy for a simple pyonephrosis is contraindicated unless ureteral drainage in these infected cases has been ineffectual and the sepsis has reached alarming proportions.

In conclusion, a review of the literature finds the majority of the authors in favor of supportive treatment, which is identically that of chronic nephritis, surgery of any type being used only as a last resort.

As far as can be determined there is no treatment for polycystic disease of the liver unless it be for one of the alarming complications which are indeed rare.

SUMMARY

1. Polycystic disease is a congenital embryological malformation affecting the kidneys, liver, and more rarely the pancreas and spleen, with a marked tendency to be hereditary.

2. The incidence of polycystic disease of the kidney in this clinic was found to be one in 5,000 admissions, and one in 228 autopsies.

a. Race and sex are equally affected.

b. It is recognized most frequently between the ages of 30 and 50.

c. Polycystic liver is frequently associated with polycystic kidney but may appear alone.

3. Important symptoms of polycystic disease of the kidney are pain in the flank or epigastrium, enlargement of the kidney, hematuria, and signs of chronic nephritis.

4. Polycystic liver rarely produces symptoms.

5. Urographic findings are very definite and characteristic, and of great diagnostic importance where the kidney is involved.

6. Diagnosis, as a rule, is not difficult with complete study.

7. Treatment is supportive, with surgery reserved for severe complications.

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The names listed above are officers for the term beginning July 1, 1939 and ending June 30, 1940.

NEW APPOINTMENTS FOR SESSION 1939–1940

Col. W. N. Bispham, Lecturer in Medicine
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Dr. William A. Parr, Assistant in Otology
Dr. Israel Rosen, Assistant in Pediatrics

DR. SAMUEL SNYDER, Assistant in Medicine
DR. CLEO D. STILES, Assistant in Diseases of the Nose and Throat

MEETING OF SOUTHERN MEDICAL ASSOCIATION

The Thirty-Third Annual Meeting of the Southern Medical Association will be held in Memphis, Tennessee, November 21 to 24, 1939.

A get-together dinner for the University of Maryland alumni attending the meeting will take place on Thursday, November 23.

Dr. L. Carl Sanders, of Memphis, class of 1915, is acting as chairman of the Dinner Committee.

The following Baltimore members of the Faculty and alumni of the University of Maryland will attend the meeting:

DR. LOUIS H. DOUGLASS
COL. W. N. BISPHAM
DR. F. A. HOLDEN
DR. HARRY C. HULL
DR. J. MASON HUNDLEY, JR.
DR. W. RAYMOND MCKENZIE
DR. J. G. M. REESE
DR. H. M. ROBINSON, SR.
DR. E. P. SMITH
DR. R. G. WILLSE
DR. LAWRENCE F. WOOLLEY

Dr. Louis H. Douglass will represent the Medical Alumni Association and discuss activities of the Medical School at the get-together dinner.

ITEMS

Dr. Clyde A. Clapp, Professor of Ophthalmology, delivered a lecture on "Treatment of Cataract" before the postgraduate group at the University of Rochester on July 25, 1939.

Dr. Christopher C. Shaw (U. of M. 1931) of Bellows Falls, Vermont, has been awarded the Certificate of the American Board of Internal Medicine.

Dr. William C. Gordon (U. of M. 1937) has been appointed assistant pathologist at the Metropolitan Hospital, Welfare Island, New York City.

Doctor Herbert Schoenrich, class of 1907, who for many years has been located on Linden Avenue together with the late Doctor Sylvan H. Likes in the practice of genito-urinary diseases, announces the removal of his office to 111 East Preston Street, southwest corner of Calvert.

OBITUARIES

MAURICE I. STEIN, M.D.

Dr. Maurice I. Stein, U. of Md. 1909, aged 52, died at Harrisburg, Pennsylvania, on August 15, 1939.

Dr. Stein was born in Baltimore, where he attended the public schools. After his graduation in medicine he interned at Sinai Hospital. During the following years he took postgraduate work in gastro-enterology in the University of Pennsylvania, Johns Hopkins School of Medicine, the Mayo Clinic, and the Mount Sinai Hospital in New York City.

During the World War Dr. Stein served in the United States Army. He was one of eight physicians chosen to study chest ailments in the various Army camps.

In 1923 he moved to Harrisburg, Pennsylvania, where he continued to practice until his death.

Dr. Stein was secretary and treasurer of the Harrisburg Hospital staff for five years, resigning in January, 1939, and was instructor of medicine and in charge of student health nursing at the Harrisburg Hospital Training School.

He was an associate of the American College of Medicine, member of the American Medical Association, Dauphin County Medical Society and the Harrisburg Academy of Medicine.

Dr. Stein was greatly interested in the Boy Scout movement in Harrisburg. In 1928 he erected and endowed a hospital at the Loysville Boy Scout Camp and acted in the capacity of advisor for the hospital.

GEORGE W. HEMMETER, M.D.

Dr. George W. Hemmeter, U. of Md. 1901, aged 63, died on July 12, 1939 at the Johns Hopkins Hospital.

At the time of his graduation from the School of Medicine of the University of Maryland, Dr. Hemmeter received the gold medal award for the highest scholastic standing in the class.

In 1904 he was appointed health warden in the Baltimore City Health Department, where he served continuously until his death. During this time he was a school medical examiner and an investigator of communicable diseases. He also served in various posts at the University Hospital, the University of Maryland and the Maryland Medical College.

For a number of years Dr. Hemmeter was professor of physiology at the Women's Medical College of Baltimore. On December 6, 1933 he became full time health officer for the City. In 1934 he was assigned to special work for the bureau and in 1935 was active in forming the Western Health District, finally being named district health officer in full charge of administration and field activities. He held this position until his death.

Dr. Hemmeter was a fellow in the American Medical Association.

COL. L. MERVIN MAUS, U. S. A., M.C., RET.

Col. L. Mervin Maus, U. of Md. 1874, aged 88, died at his home in Rockville, Maryland on August 3, 1939.

Dr. Maus was born at Burnt Mills, Maryland on May 8, 1851. He received his early education in Maryland and finally attended St. John's College in Annapolis before entering the School of Medicine of the University of Maryland.

According to the Army and Navy Journal, he entered the Army on November 10, 1874 as a first lieutenant in the Medical Corps and served until May 8, 1915. Early in his career he was awarded the Distinguished Service Medal for saving a detachment of troops from death at the hands of hostile Sioux Indians.

During the Spanish-American War his work in making captured Spanish towns fit for occupation by American soldiers reduced the ravages of disease, particularly yellow fever, among the men.

Later, as chief surgeon of American forces in Northern Luzon, Philippine Islands, in the Philippine Insurrection, he initiated a drive against the cholera epidemic of 1901–02. In recognition of this feat he was made president of the Board of Health of the Philippine Islands.

Meanwhile, Colonel Maus had been perfecting a syphilis prophylactic which was later to be adopted as standard by the Army. He was the first Army medical officer to attend the Pasteur Institute of Paris.

During the World War Colonel Maus was recalled to active duty and served from June 20, 1917 to March 1, 1919.

After the war he came to Washington and wrote many special articles on eugenics, heredity and alcohol.

Aldrich, Charles L., Paris, Maine; B.M.C., class of 1901; aged 75; died, April 28, 1939, of cerebral hemorrhage and angina pectoris.

Atkeson, Clarence Lee Crawford, Columbia, Ala.; P. & S., class of 1884; aged 78; died, July 14, 1939.

Bernstein, Edward Jay, Detroit, Mich.; class of 1887; aged 75; died, March 30, 1939, of cerebral thrombosis.

Casey, William Bradford, Norwich, Conn.; class of 1906; aged 58; died, April 12, 1939, of carcinoma of the rectum.

Causey, Peter Prentiss, Courtland, Va.; class of 1897; aged 66; died, June 5, 1939.

Comer, John Randolph, Hillsboro, Texas; class of 1892; also a minister; died, April 27, 1939.

Conarton, Joseph Leo, Mayfield, Pa.; P. & S., class of 1915; aged 55; died, May 21, 1939.

Craig, Thomas Eli, Sabino, Ohio; P. & S., class of 1892; aged 73; died, April 2, 1939, of mitral insufficiency and chronic hepatitis.

Curry, W. C., Flemington, W. Va.; class of 1881; aged 85; died, March 25, 1939, of chronic myocarditis and arteriosclerosis.

Dundon, Arthur Hall, North Plainfield, N. J.; B.M.C., class of 1900; aged 62; died, June 20, 1939, of chronic arthritis.

Edmunds, George Franklin, Bristol, Vt.; B.M.C., class of 1892; aged 76; died, March 18, 1939, in the Bradenton (Fla.) General Hospital, of arteriosclerosis.

Felts, Robert L., Durham, N. C.; class of 1898; aged 68; died, May 27, 1939, of coronary heart disease.

Flowers, Frank E., Mannington, W. Va.; P. & S., class of 1907; aged 55; died, March 31, 1939, of angina pectoris.

Gibbons, Edward Engler, Baltimore, Md.; aged 67; died, May 30, 1939, of acute myocarditis.

Hayes, John Mortimer, Davidsonville, Md.; class of 1901; aged 64; died, May 16, 1939, of arteriosclerotic cardiovascular disease.

Henning, Emil Heller, Baltimore, Md.; class of 1908; aged 58; died, March 4, 1939, of Hodgkin's disease.

Hoff, David Edward, Harrisburg, Pa.; class of 1902; aged 64; died, May 31, 1939, of left ventricular failure and cerebral embolism.

Hope, William Davis, Lockhart, S. C.; class of 1887; aged 73; died, April 27, 1939.

Hunter, Willis Monroe, Van Wert, Ohio; B.M.C., class of 1892; aged 72; died, May 21, 1939, of cerebral hemorrhage.

Jackson, Byron Hubbard, Scranton, Pa.; B.M.C., class of 1898; aged 65; died, May 16, 1939, in the Moses Taylor Hospital.

Keister, Bittle Cornelius, Washington, D. C.; P. & S., class of 1882; aged 82; died, May 3, 1939.

Kelly, John Clarence, Mitchell, Ind.; P. & S., class of 1882; aged 80; died, March 10, 1939, in the Homeopathic Hospital, Wilmington, Del., of carcinoma of the tonsil with abdominal metastasis.

LaFortune, Wilfred T., Fitchburg, Mass.; B.M.C., class of 1910; served during the World War; aged 52; died, May 8, 1939, of coronary thrombosis.

McComas, Henry Wheeler, Oakland, Md.; class of 1888; aged 74; died, April 10, 1939, of carcinoma of the prostate with metastasis.

McIntyre, Milton Underwood, Dubois, Pa.; B.M.C., class of 1907; aged 56; died, May 25, 1939, of coronary thrombosis.

Paris, Oliver J., Graham, N. C.; B.M.C., class of 1897; aged 72; died, May 2, 1939, of strangulated hernia, chronic nephritis and myocarditis.

Parker, Leonard Murray Campbell, Baltimore, Md.; B.M.C., class of 1900; aged 60; died, April 22, 1939, in the Mercy Hospital of arteriosclerotic heart disease.

Pegram, Robert W. S., Canton, N. C.; B.M.C., class of 1892; served during the World War; aged 74; died, March 26, 1939, at Augusta, Ga., of myocarditis.

Post, W. H., Masontown, W. Va.; B.M.C., class of 1901; aged 61; died, March 18, 1939, in the Monongalia County Hospital, Morgantown, W. Va.

Reed, John Henry, Logansport, Ind.; class of 1885; aged 78; died, March 2, 1939, of pneumonia.

Robertson, Robert River, Portsmouth, Va.; P. & S., class of 1887; aged 77; died, May 24, 1939.

Rogers, Edward Bancroft, Collingswood, N. J.; P. & S., class of 1903; aged 61; died, May 12, 1939, of pneumonia.

Smouse, David Wilson, Los Angeles, Calif.; class of 1876; aged 85; died, July 2, 1939, of arteriosclerosis.

Stewart, W. B., Georgetown, W. Va.; P. & S., class of 1905; aged 76; died, May 29, 1939, of intestinal obstruction.

Studebaker, George M., Erie, Pa.; B.M.C., class of 1896; aged 72; died, March 9, 1939, of cerebral hemorrhage and arteriosclerosis.

Turner, John F., Canton, S. D.; B.M.C., class of 1893; aged 72; died, June 21, 1939, following an operation of the prostate.

Vieweg, Max W., Wheeling, W. Va.; class of 1917; aged 55; served during the World War; died, August 13, 1939, of pulmonary tuberculosis.

Ward, Columbus C., Crisfield, Md.; B.M.C., class of 1894; aged 74; died, May 26, 1939, in the University Hospital, Baltimore, of carcinoma of the pancreas.

Williams, A. R., Aiken, S. C.; B.M.C., class of 1892; aged 68; died, March 14, 1939, of coronary thrombosis.

Windley, Richard Eugene, Vanceboro, N. C.; class of 1903; aged 61; died, April 25, 1939, of coronary thrombosis.

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THE NEW FRANK C. BRESSLER RESEARCH LABORATORY

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UNIVERSITY OF MARYLAND

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EDITORIAL

The Frank C. Bressler Research Laboratory of the School of Medicine, University of Maryland

Silhouetted against the hazy autumn sky, casting its shadow over the Pantheon building immortalized by Lafayette and McDowell, the new Bressler laboratory adds grace and dignity to the venerable campus of the University. It amalgamates the glorious record of the ever-lengthening past with its dynamic present and portends a vigorous growth for the future of the institution.

The Bressler Research Laboratory is completed. Within a month the surly clang of the air hammer will be replaced by the synchronous click of the kymograph and the awe-inspiring silence of medical research. During the Christmas vacation period many of the preclinical departments will take up their new commodious quarters in the Bressler building, with every modern facility for teaching and research. The formal dedication of the building will be held during the alumni week in May.

The Bressler Research Laboratory is a six story red brick building of modern architecture with limestone trim and resembles the new University Hospital which it faces on the other side of Greene Street. The building extends eastward from Greene Street 186 feet and northward from the old Gray Laboratory building 63 feet. The building is shaped like the letter I. Some of the special features of the structure are the stainless steel outside doors, the ornate lobby of golden travertine and black Tennessee marble, and the terrazzo floor and cinder block partitions.

The first floor of approximately 11,000 square feet has on it the Bressler Memorial Room, beautifully decorated, which is to be used for a students' lounge. The remainder of the floor, comprising about

9,000 square feet, houses the Department of Gross Anatomy under the direction of Professor Eduard Uhlenhuth. The east end of the first floor is devoted to the students' work in dissecting. The dissecting laboratory is modern and efficient in every respect, with its white tile floor, batteries of white enamel wash basins, Monel metal dissecting tables and shadowproof illumination. In addition there are several well-equipped research laboratories, including space devoted to the teaching of living anatomy, and well-appointed professorial offices.

The second floor, of the same area as the first, is devoted to Embryology and Histology under the directorship of Professor Carl L. Davis. The spacious students' laboratory is furnished with the most modern desks for microscopic study. The department is adequately provided with individual research rooms, an embryological museum and a constant temperature room. On the same floor is a lecture hall with all modern appointments.

The Department of Pharmacology under the direction of John C. Krantz, Jr. will occupy the third floor. A commodious students' laboratory for mammalian experimentation is situated in the east end of the floor. Twelve well-equipped research laboratories, animal quarters and a modern operating room occupy most of the remaining space on this floor. A feature of this department is a fully equipped laboratory of organic chemistry for the synthesis of new compounds likely to exhibit pharmacologic activity. Besides, the third floor will house the departmental libraries of the occupants of the Bressler building in a beautifully appointed library and seminar room.

The fourth floor will be devoted to the Department of Physiology under the direction of Professor William R. Amberson. The students' laboratory is excellently equipped for amphibian and mammalian work. A metabolism room built in the main students' laboratory is a feature of the fourth floor plan. The professorial offices and research rooms are spacious and well-equipped. Worthy of special mention is the laboratory devoted to oscillograph studies under Assistant Professor Robert H. Oster. A laboratory for roentgenological studies has also been provided.

The photograph of the building shows a diminution in size of the floors above the fourth. The I-shaped building becomes rectangular. The fifth floor of approximately 9,000 square feet houses the research divisions of the clinical departments in addition to a splendidly equipped students' laboratory for clinical pathology. Under the direction of Professors Pincoffs, Shipley, Douglass and Hundley and the heads of other subdepartments of the clinical subjects, there are ten well-equipped research units which include operating rooms, chemical laboratories and rooms for specialized biological studies. There is also a sizable seminar and conference room on this floor where a departmental library will be organized.

The sixth floor houses the quarters for animals and the students' laboratories for dog surgery and cadaver surgery under the directorship of Clinical Professor Aycock and Assistant Professor Yeager, respectively. The animal quarters were designed largely by Professor Amberson and have already attracted wide and favorable comment because of some of the novel features that have been instituted for the comfortable housing of laboratory animals.

The occupants of the Bressler Research Laboratory are happy to enter their new quarters, as it is the fruition of dreams and hopes of a generation ago. To them falls the obligation to extend within these walls the frontiers of knowledge for the alleviation of pain and suffering.

Indeed, a great debt of gratitude is due the distinguished alumnus, Dr. Frank C. Bressler, of the Class of 1885, whose generosity has made possible this building. His work is done. "If ye would seek his monument, look about you." Those who enter, like the Apostle Paul, realize that they are debtors to the Greeks and to the Barbarians, to the wise and to the unwise—to extend the boundaries of the medical sciences.

JOHN C. KRANTZ, JR.,
Chairman, Building Committee.

RESIGNATIONS

The following resolution was passed by the University Hospital Council at its meeting on December 19, 1939:

JAMES M. H. ROWLAND, M.D., Sc.D., LL.D.

Whereas: Dr. J. M. H. Rowland has been Dean of the School of Medicine since 1916, a member of the Hospital Council since its organization, and for many years its Chairman; and by reason of his intimate knowledge of the affairs of the School and Hospital, his incisive intellect, his fundamental honesty and forthright expression of opinion, he has rendered service of signal value to the University Hospital and its professional staff; now with the passing of time, having reached the age of retirement as Dean of the School of Medicine, and likewise from membership in the Hospital, therefore,

Be It Resolved: That the Hospital Council at its regular meeting, December 19, 1939, takes this opportunity of expressing its thanks and appreciation of his valuable service to the University Hospital and particularly to his helpfulness and unfailing courtesy in the deliberations of this Council, and wishes to extend to him its felicitations for continued good health and happiness.

The following minutes were adopted by the University Hospital Council at its meeting on November 21, 1939:

ARTHUR J. LOMAS, M.D., C.M., D.P.H.

The Hospital Council has heard with regret that Dr. Arthur J. Lomas, the Superintendent of the University Hospital and a member of this Council, has tendered his resignation to the President of the University.

Dr. Lomas came to the University Hospital in May, 1923, at a time when this institution, poorly housed in old and run-down buildings, was at a critical period in its career. The successful reorganization of the hospital service and, in time, the building of the splendid edifice in which the hospital is now housed, are largely due to his continued and devoted efforts.

As Superintendent of the hospital Dr. Lomas has combined many valuable characteristics. His interest in and extensive acquaintance with architecture and construction have been of the greatest value during the period of expansion of our facilities. The plan of the new hospital was largely the result of his gifts in this field. His sympathy with the aims of the heads of the departments in improving the level of the professional work in the institution has meant that at all times he would strain the hospital's resources in order to furnish these departments with necessary equipment and personnel to advance their work. Dr. Lomas' wide acquaintanceship in the City of Baltimore and in other parts of the country has given to him an experience in dealing with people which has enabled him to represent the hospital in its relations with other institutions in this city and elsewhere in a way very much to our advantage.

As a member of this Council Dr. Lomas has sat with us monthly since the first year of his arrival, and together we have discussed the innumerable difficulties of finance and administration which are necessarily a part of the progress of a large institution. His withdrawal from these activities will leave an unfilled place in our ranks. The Council wishes to record both its deep regret at Dr. Lomas' departure and its sincere good wishes to him for success and happiness in his new undertakings.

TULAREMIA*†

A DIAGNOSTIC AND THERAPEUTIC STUDY

FRANCIS G. DICKEY, M.D.

BALTIMORE, MD.

Not many years have passed since the first case of tularemia was diagnosed in the University Hospital. In the winter of 1928 a worker in the Department of Pathology at this hospital, while handling rabbits became infected and developed a typical picture of the disease. Since then there has been a total of twenty-eight cases recorded in the files of the University and Mercy Hospitals. During this period there have arisen many problems of diagnosis and numerous methods of treatment have been employed. It has, therefore, seemed of interest to present an analysis of this group of cases seen at the above hospitals.

Tularemia has only in recent years been recognized as a definite clinical entity. The work that led up to the formal presentation of the disease by Francis (1) in 1921 was begun in 1911–1912 by McCoy and Chapin (2), when the organism was first isolated from ground squirrels. Wherry and Lamb (3) cultivated *Bacterium tularense* from á human being (conjunctiva) in 1914. They found the organism in infected rabbits and warned of the danger of transmission of the infection to man. In the following years, Francis conducted his monumental work leading up to his naming the disease *Tularemia.* Since then many workers have added to our knowledge and reference will be made to their findings.

Annual and seasonal incidence. The yearly distribution of the twenty-eight cases can be seen from the following table:

	1928–1929	1929–1930	1930–1931	1931–1932	1932–1933	1933–1934	1934–1935	1935–1936	1936–1937	1937–1938	1938–1939
Cases......	1	1	3	1	3	1	4	5	1	1	7

There is only a vague suggestion of any increase in the number of cases year by year.

In this series, every infected individual had had some contact with rabbits, although one case had also been in contact with a muskrat. The seasonal incidence of the disease corresponded closely with the

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* From the Department of Medicine, School of Medicine, University of Maryland.

† Received for publication December 19, 1939.

season of contact with wild rabbits, i.e., the open season for this type of game. All of the author's cases were seen in either November, December or January. The hunting season for rabbits in Maryland extends from November 15 to December 31. Four cases occurred in November, eighteen in December and six in January.

In the entire series there were two deaths, giving a mortality rate of 7.1 per cent.

Diagnosis. In the vast majority of cases tularemia is not difficult to diagnose. The seasonal prevalence and the history of contact alone will suffice many times to point to the diagnosis. Many of the instances of missed diagnosis are caused by failure to obtain a history of contact with the infecting agent. All of the cases in the present group were infected through the handling or cleaning of rabbits, and of the twenty-eight cases, nine either cut their finger or stuck it with a bone spicule at the time of the cleaning. This latter fact has no definite clinical value because it is well known that the infection can pass through the unbroken skin. In this series there was no instance in which glandular involvement appeared without an accompanying lesion of the hand. In several of these cases the ulceration appeared only as a small abrasion at the base of the nail. In two cases, however, the lymphadenitis appeared before the primary lesion became significantly visible, and this point should be considered in the diagnosis.

Since tularemia may be transmitted by vectors other than rabbits, it is not difficult to see why it may be erroneously diagnosed. Mentioned in the literature as agents of transmission are flies, ticks, autopsied laboratory animals, tree squirrels, cats, dogs, quail and, less frequently, other small animals. The disease has been mistaken for influenza, septic infection, typhoid, sporotrichosis, undulant fever, and tuberculosis.

Agglutination tests are a distinct aid in diagnosis. In the first week of the disease agglutinins are completely absent, but always make their appearance during the second week and reach the highest titer during the third. This was found to be true in all cases of the present series whenever the exact time of infection could be determined. With the exception of an occasional cross agglutination of Bacterium tularense and the Brucella organisms, the agglutination reaction for tularemia is highly specific.

Foshay (11) devised an intradermal diagnostic test, using a chemically detoxified bacterial suspension. This was found to be the earliest available diagnostic aid for determining the presence of tularemia. Positive reactions have occurred as early as the fourth day of the illness, practically one week before agglutinins usually appear in the blood. This test should be made use of in doubtful cases. The skin response is specific and reliable. It remains positive for an undetermined period following the initial infection. Agglutinins likewise persist for many years in the blood of patients having had the disease. Foshay (12) feels that there is evidence that the B. tularense itself may remain in the tissues for many years in a viable state but is held in check by the defense mechanisms of the body.

In all of the cases of the present series, except one, the blood culture was negative. The routine blood agar and brain broth media were used and, as is now well known, the organism grows very poorly or not at all on these media. The positive culture was obtained in an overwhelming infection. This occurrence has likewise been reported by Lufkin and Evenson (13). The organism can be grown in media containing cystine and this should be used when tularemia is a probability.

Clinical types. It has been the custom in the past to subdivide tularemia into the following clinical types:

1. Ulceroglandular
2. Oculoglandular
3. Glandular
4. Typhoid
5. Ingestion
6. Pneumonic

To these may be added a meningitic type and a tularemic angina. Apparently, whenever a new system or part of the body is found involved, a new type is added to the classification. A more fundamental classification is needed and this has been suggested by Reimann (4). He noted that tularemia, like plague, occurs in three clinical forms: bubonic or glandular, pneumonic, and septicemic or typhoid. He stated that the causative bacteria of plague and tularemia are closely related, similar animal species serve as reservoirs of infection, and both diseases may be conveyed to man by direct contact with infected flesh or blood, by inhalation, and by insects. Francis saw the similarity between the infection as he found it in animals and the plague-like disease of rodents as had been previously described. The frequency with which pneumonia is found in cases of tularemia brings out a further resemblance between this disease and plague. The advisability of using this simpler classification,

1. Glandular (bubonic)
2. Septicemic (typhoid)
3. Pneumonic

should be considered. In further consideration of this thought, the pneumonic type could even be considered as a complication of the septicemia and thus be eliminated from the classification. This would leave merely the two: a more local type, the glandular, and a general systemic type, the septicemic.

Incubation time. It was possible to learn the exact time of the incubation period in twenty-one of the present series of twenty-eight cases. The average was four days, but the data on certain cases indicated that the incubation period is occasionally only one or two days, that is, the disease is very swift in its onset in some instances. Francis states that the average is three and one-tenth days (5).

Examples of the three types of the disease, glandular, pneumonic and septicemic, were observed in this series.

Glandular type. The glandular or bubonic type was by far the most common. Twenty-five of the twenty-eight cases were of this type. There was no selection of age, sex or race in this class. The symptoms were very similar in all cases, with severe headache usually predominating. There was noted malaise, repeated chills associated with fever, profuse sweating, anorexia, and in several of the cases a slight cough. The prostration of these patients was noteworthy.

The lesion first seen was usually a small ulcer on one of the fingers, beginning as a painful, inflamed papule or pustule which liberated a necrotic plug. Following this primary lesion, an inflammatory process is set up in the lymph glands which drain the area, resulting in the firm, tender, and at times quite painful glands seen in the axillary or epitrochlear regions. Twenty-two of the cases showed axillary involvement whereas only thirteen showed epitrochlear. Usually the physical examination was negative in other respects, although there were a few moist rales found in those that had a cough.

There has been noted occasionally an associated skin eruption and three of these cases showed this, it being present as a bright red papulopustular lesion with a few vesicles and eventually scaling. It may appear in other forms and in itself does not aid in diagnosis. It was present in the author's cases on the arms and neck; it has also been reported as occurring on the trunk and abdomen.

The disease was accompanied in all cases by fever ranging between 101° and 103° and septic in character, as a rule. There was only a moderate increase in pulse rate to about 90 and practically no increase in the respiratory rate. The white blood count averaged 10,000, the extremes ranging from 4,000 to one case with 22,000. The differential count tended to show a small increase in the number of lymphocytes, though not enough to be of any diagnostic value. Urinalysis was essentially negative, except for an occasional case exhibiting a trace of albumin. There was no evidence of anemia. The routine blood cultures in every case except one were negative.

The average duration of fever was between two and three weeks, with a gradual return to normal by lysis. A mild secondary rise in temperature was noted in one case, and this feature has been reported frequently in the literature.

Septicemic type. In the entire series of twenty-eight cases there was only one of the septicemic type of tularemia. This occurred in the past year and was in a white male, thirty-eight years old, who worked for a produce company where he handled dead rabbits. He developed typical symptoms of the disease and on admission to the hospital the diagnosis was suspected. There were no lesions of any kind either on his hands, upper arms, or elsewhere. A positive agglutination was obtained in a dilution of 1:320. He likewise ran a course of two and one-half weeks, which terminated with lysis. His white blood count never went above 6,800, and there was a slight increase in the number of lymphocytes. It is possible that the moderate leukocytosis found in many of the other cases may have resulted from the secondary infection present in the open wounds of the ulcerating type. Beck and Merkel's (6) fatal case of tularemia, caused by the ingestion of improperly cooked rabbit and exhibiting intestinal ulcers, will fit into this class.

Pneumonic type. The third type of tularemia, the pneumonic, was present in four of the twenty-eight cases. As a typical example, the case previously reported by Gundry and Warner (7) will be described. It is that of a white male, aged fifty-three, who early in November killed and skinned some rabbits. Two days later he began having sweats, fever and great prostration. A diagnosis of "grippe" was made. His illness progressed and in a week he had a severe cough with pain in his chest, and was told he had pneumonia. Twelve days after his illness began he was admitted to the hospital. Examination showed that he was acutely ill, quite cyanotic, and dyspneic. His skin was hot and dry, and he was delirious. On examining the chest it was seen that the expansion of the right side was limited and the percussion note was impaired. There were numerous coarse rales over the entire chest, most marked at the right base. The heart was negative except for tachycardia. His abdomen was distended and tender throughout. There were several confluent ulcers on the dorsum of the right hand, several glands in the right epitrochlear region and one large gland in the right axilla. Urinalysis showed a 3 plus albumin, an occasional granular cast, white blood count of 4,000, with 78 per cent polymorphonuclears, 20 per cent lymphocytes, and 2 per cent transitionals. Agglutination for tularemia was positive in a dilution of 1:800. From the patient's blood a growth of Bacterium tularense was obtained. His course was steadily downhill, and respirations ceased twenty-four hours after admission.

Postmortem examination of this patient showed an extensive bronchopneumonia, plus small necrotic nodules characteristic of tularemia in the liver and spleen. Material from the necrotic foci in the spleen was macerated and inoculated subcutaneously in a guinea pig. This animal died in five days with the typical visceral lesions of guinea pig tularemia.

The importance of keeping this type of case in mind becomes apparent when one realizes the frequency of pulmonary complications in tularemia. In 1933, Gudger (8) reported that 92 per cent of fatal cases of tularemia showed intrathoracic lesions of tularemia, and 61.5 per cent showed definite pneumonia. Two years later Blackford (9) described the clinical types of the pulmonary manifestations. He divided them into pneumonia, bronchitis and pleural effusion. He felt that the chest lesions simply followed as complications of the usual primary lesions or were a part of a septicemic invasion. Winter, Farrand and Herman (10) reported four cases of pulmonary lesions with recovery in all cases, but in none of their cases were ulcerating skin lesions or glandular involvement present. The author's cases tend to coincide with those of Blackford, because in three of the four cases there was primary ulceroglandular involvement. Two of these four cases died as a result of the overwhelming pulmonary infection. Although the seriousness of tularemic pneumonia is not to be denied, the present tendency is to consider it less fatal than previously recorded. Early treatment with antiserum has helped produce this change of opinion. Winter et al. noted that following recovery from tularemic pneumonia, dyspnea on exertion and general weakness were present for some months; they felt that a toxic myocarditis was a usual sequel.

Any atypical pneumonia with a relatively low white blood cell count, a slow pulse rate, and slow evolution of lung changes should cause one to suspect tularemia. This disease must also be considered in cases of pleurisy with effusion. A chest roentgenogram should be taken in every case of tularemia.

Treatment. As a prophylactic measure rabbits should be cooked thoroughly, so as to render the flesh of an infected rabbit harmless. Tularemia can be acquired through the ingestion of improperly cooked food. Rubber gloves should be worn by cooks, market men and hunters while dressing wild rabbits, and by laboratory workers while performing autopsies on infected animals.

The chief treatment of a case of tularemia is symptomatic and supportive.

During the past ten years several specific therapeutic agents have been advocated, but none has been thought by everyone to be the best. Probably the most successful is Foshay's antitularemic serum (14). This is a specific antiserum prepared from a goat or horse inoculated with a formaldehyde killed suspension of Bacterium tularense. This was used in six of the author's cases, with one death. This fatality was in a patient who had been ill with the disease for sixteen days before admission to the hospital; the serum was given on the seventeenth day, and death occurred on the nineteenth day. The other cases seemed definitely to have been benefited by its use. It is advised to give about 15 cc. of the serum intravenously on two successive days. Later workers (15) have used quantities of 30 cc. the first day and 90 cc. the second day. The author's experience encourages him to employ the serum in all seriously ill patients.

An autogenous vaccine made from organisms grown from the involved glands has been employed. This was tried in one of the author's cases, a progressively increasing dose being given subcutaneously. The result was again good, but whether the vaccine itself caused the response cannot be said from this one case.

Neoarsphenamine (16) was formerly advocated but it has fallen into discard. More recently Elson (17) reported using ferrous iodide, and he considers it a specific. Sulfanilamide was given to one of the cases in this series, with no effect. With sulfapyridine, the same patient's temperature showed a slight drop, but because of nausea and vomiting the drug had to be discontinued. There has been a recent report of favorable results from the use of sulfanilamide (18).

In previous years irradiation of the cutaneous lesions was advocated. The author has used this treatment in several cases, with little or no beneficial effect. It is said to be of value in hastening resolution of the lymphadenopathy. In one-third of the cases it was ultimately necessary to incise and drain the glandular abscesses.

SUMMARY

Twenty-eight cases of tularemia occurring in the University and Mercy Hospitals are analyzed. The important points in the diagnosis are found to be seasonal prevalence, history of contact with rabbits, an ulceroglandular lesion, headache, feverishness, and great prostration. The frequency of pulmonary complications is discussed and the possibility of atypical pneumonias caused by Bacterium tularense mentioned.

The blood agglutination test was found to be quite accurate, but the intradermal test affords a means of earlier laboratory diagnosis.

The therapeutic value of antitularemic serum is emphasized and experience with the newer chemical agents is reported.

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THE HYPERACTIVE CAROTID SINUS REFLEX*†

Report of Two Cases

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The purpose of this article is to describe a disorder which only recently has been introduced into medical literature as a clinical entity, and to emphasize the importance of considering the hyperactive carotid sinus reflex in obscure cases of syncope and peculiar types of convulsive seizures.

To Jacques Roskam (7) must go the credit for originally describing in 1930 a case of repeated syncopes caused by hyperreflexia of the carotid sinus. For the physiological explanation of his case he gave credit to the publications of H. E. Hering (4) on the role of the carotid sinus in the regulation of the arterial tension.

In 1933, Soma Weiss and J. P. Baker (11) described the carotid sinus reflex in health and disease, and discussed its role in the causation of fainting and convulsions. Since that time a number of papers have been written describing various abnormalities of the carotid sinus reflex.

The carotid sinus is the bulbous dilatation of the common carotid artery at the point of its bifurcation into the internal and external carotid arteries. This area, beside containing the carotid gland (glomus caroticum), is also richly supplied with a plexus of nerve fibers from which originate the carotid sinus reflex. The afferent paths of the reflex are by way of the cervical portion of the sympathetic chain and the intercarotid, glossopharyngeal, hypoglossal and vagus nerves. The stimuli traverse central synapses in the brain stem and innervate contralateral autonomic pathways, both central and peripheral, in a variable manner. It is these variants in the efferent pathways that produce the different types of responses.

Weiss and his coworkers have classified these responses into three groups: vagal, depressor and cerebral. Though these types may occur singly in various individuals, there is frequently a combination of all three types of responses in the same individual, with one of the three types predominating. The vagal type is characterized by slowing of the pulse and even asystole. This slowing of the pulse occurs within a few seconds after stimulation of the carotid sinus is begun, with a return to the normal pulse rate within twenty to thirty seconds, even with continued stimulation of the sinus. The depressor type is characterized by a drop in the systolic and diastolic blood pressures. The cerebral type is manifested by syncope and in some cases convulsions.

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* Published with permission of the Medical Director, Veterans Administration Facility, who assumes no responsibility for the opinions expressed or conclusions drawn by the authors.

† Read before the Richmond County Medical Society, Augusta, Georgia, March 16, 1939.

Carotid sinus syncope occurs much more commonly in males than in females. Harry L. Smith (8) found in a series of eighty-five cases that the ratio of males to females was 5:1. It is most common among middle-aged and elderly individuals. The youngest case reported was in a child of twelve years and the oldest in a patient seventy-eight years of age. The average is about fifty-six years.

The following disorders have been mentioned as etiological factors in producing spontaneous attacks of abnormal activity of the carotid sinus reflex: tumors of the neck (particularly of the carotid body), cervical adenitis, aneurysmal dilatation of the carotid sinus, arteriosclerosis, and syphilis of the central nervous system. Attacks may occur spontaneously following a sharp twist of the neck, by stretching the skin of the neck as in shaving, by the wearing of tight collars, and by overexertion and emotional disturbances. In many cases no cause for the attack can be ascertained.

The attacks usually take place when the patient is in an upright position and are relieved if he lies down when premonitory symptoms occur. A definite aura is usually present. This consists of one or more of the following: dizziness, weakness, hyperpnea, lightheadedness, spots before the eyes, epigastric distress, facial pallor, numbness and tingling in the extremities, profuse perspiration, nausea, cough, lacrimation and palpitation. If the attack is mild, the aura is followed by vertigo and the patient staggers and may fall if he does not quickly reach a support. When the attack is more severe, the patient falls to the ground unconscious and unconsciousness lasts, on the average, from a few seconds to five minutes. One case has been reported in which unconsciousness lasted as long as one hour. Incontinence of the bowel and bladder rarely occurs. Following the attack the patient usually feels weak for several hours and suffers from headache or, in rare instances, from continued dizziness.

Clinically, detection of a hyperactive carotid sinus reflex is carried out in the following manner. The patient is placed in a sitting position, and blood pressure estimations and pulse rates are taken until a basal level is reached. The carotid sinus is ordinarily located at the anterior border of the sternomastoid muscle at the level of the upper border of the thyroid cartilage. Palpation of this area reveals the sinus at the point of maximum pulsation. In some individuals the sinus is located lower than the upper border of the thyroid cartilage, and in others it may be found behind the angle of the mandible. When locating and massaging the sinus, the patient's head is turned to the opposite side. One examiner places his thumb firmly upon this area and compresses it against the cervical vertebrae, massaging the sinus for thirty seconds; the other examiner meanwhile takes pulse and blood pressure readings. Of course, only one sinus is stimulated at a time. If a reaction is obtained, it is necessary to wait for a return to the basic pulse and blood pressure readings before stimulation of the other sinus is attempted.

Most individuals who show hyperactivity of the carotid sinus reflex are hyperactive bilaterally, but with a preponderance of symptoms and findings on only one side. Stimulation of a hyperactive carotid sinus by massage characteristically produces a drop in pulse rate and of blood pressure, and cerebral symptoms as previously mentioned. The cerebral symptoms vary from weakness and dizziness to actual unconsciousness and correspond exactly with the symptoms experienced during a spontaneous attack.

Instillation of 1 per cent procaine hydrochloride in the region of the carotid sinus abolishes those reactions which are not psychogenic in origin. Digitalis sensitizes, and atropine and sympathomimetic drugs inhibit the carotid sinus reflex. The reflex may also be completely abolished in some instances by surgical extirpation of the plexus, which is carried out by stripping the outer coat of the common carotid artery for 2.5 cm. below, and the internal and external carotid arteries for 2.5 cm. above the bifurcation, with removal of the tissue including the carotid body.

Stimulation of the carotid sinus has no effect in cases of narcolepsy, catalepsy, petit mal and grand mal, although cases of hyperactive carotid sinus reflex have been described in which convulsive seizures almost mimic the attacks of grand mal.

Treatment of carotid sinus hypersensitivity with drugs has not proved particularly satisfactory. The drugs usually recommended are ephedrine and epinephrine for the pure depressor type, and atropine for the pure vagal type. Recently benzedrine has been used, but with little success. The exhibition of drugs is of little avail in vagal-cerebral, depressor-cerebral, or pure cerebral types. The treatment of choice in these types, and indeed in all cases of hypersensitive carotid sinus reflex, is surgical denervation, as above described.

CASE REPORTS

No. 1. G. H., a white male, age 42 years, was admitted to the Veterans Administration Facility, Augusta, Georgia on November 26, 1938. His chief complaint upon admittance was "dizzy spells". The family history was essentially negative except that the entire family was said to be of a "nervous type". One brother, age 38 years, was described as feebleminded. The past history also was essentially negative, with the exception of the finding of a two plus blood Wassermann in 1923. This finding was not corroborated on subsequent examinations and treatment for syphilis was never instituted.

The onset of his present illness dated back to 1922, when the patient first experienced a peculiar spell of dizziness which was induced for no apparent reason. Since that time the patient experienced similar episodes about twenty times each year. He stated he knew no definite reason or cause that brought on these attacks except, perhaps, overexertion.

Description of attack. The patient had definite premonitory warnings or an aura of an impending attack by noticing shortness of breath, dizziness, a feeling that his head was swelling, a ringing in his ears, blurring of vision, and epigastric distress. If the attack was a mild one and he could reach a support or a bed in time, the attack would pass off within a few minutes without occurrence of unconsciousness. However, the aura was often immediately followed by unconsciousness, during which he fell to the ground. He had been told that he remained unconscious for two or three minutes but had no incontinence of the bowel or bladder and no convulsive seizure. Following the period of unconsciousness, he felt weak and dizzy for several hours afterwards. He also noticed after this period of unconsciousness marked sweating of his hands and numbness and tingling in his fingers.

Physical examination. The patient was a well-developed, well-nourished, white male, 42 years old, height 70¾ inches, weight 159 pounds. The head and neck were essentially negative, with the exception of a three-quarter inch scar just beneath the midpoint of the body of the right mandible. Examination of the cardiorespiratory system revealed the heart borders well within normal limits; heart sounds of good quality; no murmurs; no arrhythmia; no thickening, beading, or tortuosity of the peripheral vessels; blood pressure 136 mm. of mercury systolic and 82 diastolic; pulse rate 68 per minute, after exercise 84 per minute; lung fields entirely negative. Abdominal examination showed an old scar of appendectomy and a relaxed right inguinal ring. The genitalia and rectum were negative. Extremities were negative.

Laboratory data. Urinalysis was negative throughout. The blood picture was as follows: erythrocytes 4,802,600; hemoglobin 80% (Tallqvist); leukocytes 7,600; polymorphonuclears 61%; lymphocytes 26%; monocytes 11%; eosinophils 2%. The blood Wassermann test was negative. Spinal fluid: globulin negative; 4 cells per c.mm.; Wassermann test negative; Lange colloidal gold test negative.

The neurological examination was negative throughout, with the exception of slightly hyperactive deep reflexes.

Mental examination was entirely negative, with the exception of some slight impairment of the memory for recent and remote events. The patient showed no evidences of suggestibility nor of a psychoneurosis.

Special investigation of the carotid sinus reflex. Stimulation of the left carotid sinus by massage caused the blood pressure to drop from 136/76 to 120/70. The pulse rate dropped from 82 per minute to 76 per minute. However, there were no subjective symptoms, no dizziness, and no unconsciousness consequent to the stimulation of the left carotid sinus. Massage of the right carotid sinus caused the blood pressure to drop from 132/78 to 98/66; the pulse rate dropped from 88 per

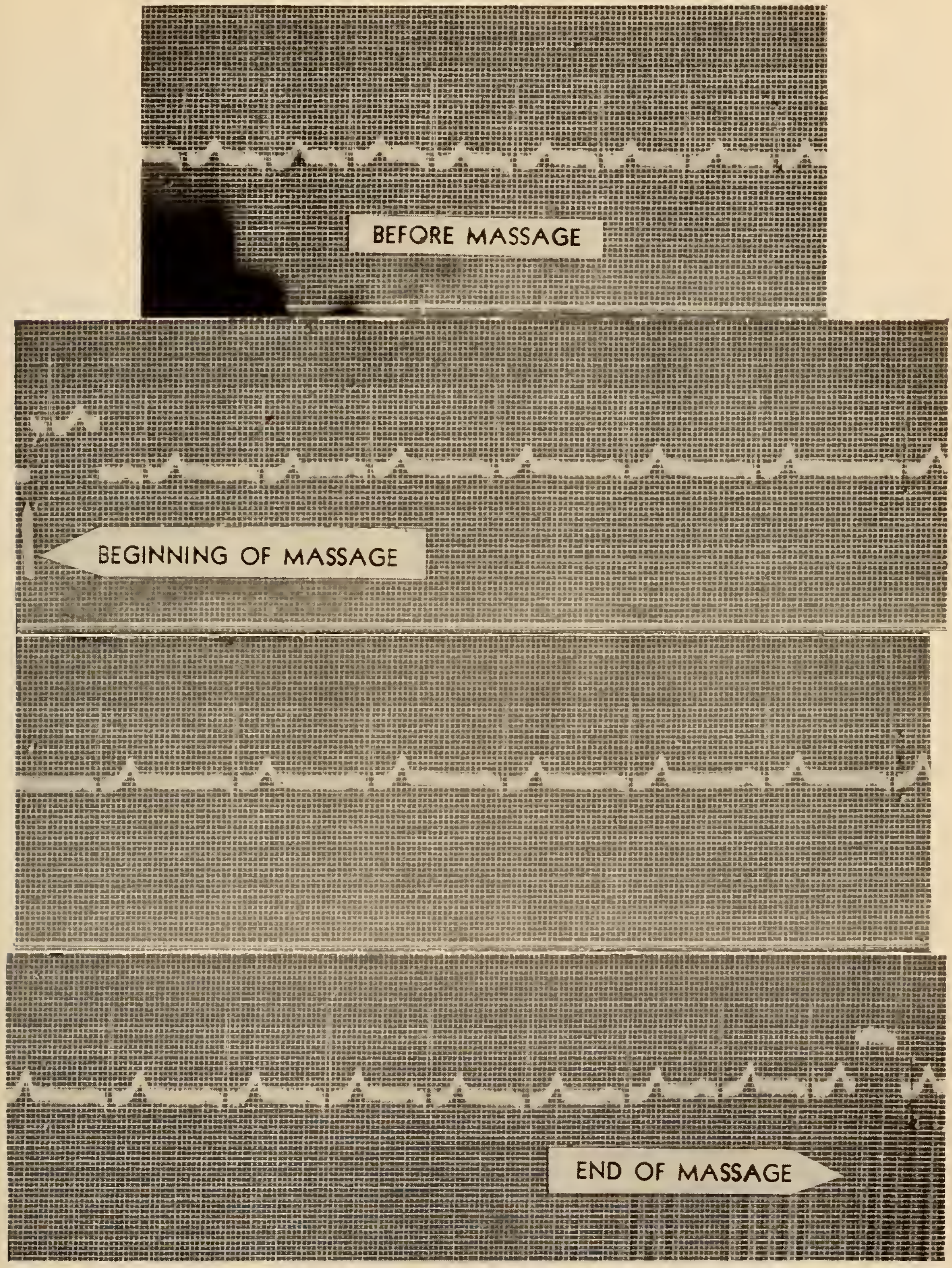

Fig. 1. Case No. 1. Electrocardiogram taken during massage of right carotid sinus.

minute to 60 per minute, and after twenty seconds of massage the patient lapsed into a brief period of unconsciousness, his face became pale, and his breathing deep and rapid. Upon recovering consciousness a few seconds later the patient stated that he had all the subjective symptoms that were present during a spontaneous attack. The test was repeated a number of times with identical results. The drop in the pulse rate was noted within a few seconds after massage was begun, and had returned to its previous level within thirty seconds though massage was continued during this period of time. The electrocardiogram in Figure 1 demonstrates the marked slowing of the pulse during stimulation of the right carotid sinus. Suggestibility on the part of the patient was ruled out by massaging the common carotid artery below the sinus, whereby neither subjective nor objective symptoms were produced. It is noteworthy that palpation of the right carotid sinus caused much more pain than did palpation of the left sinus.

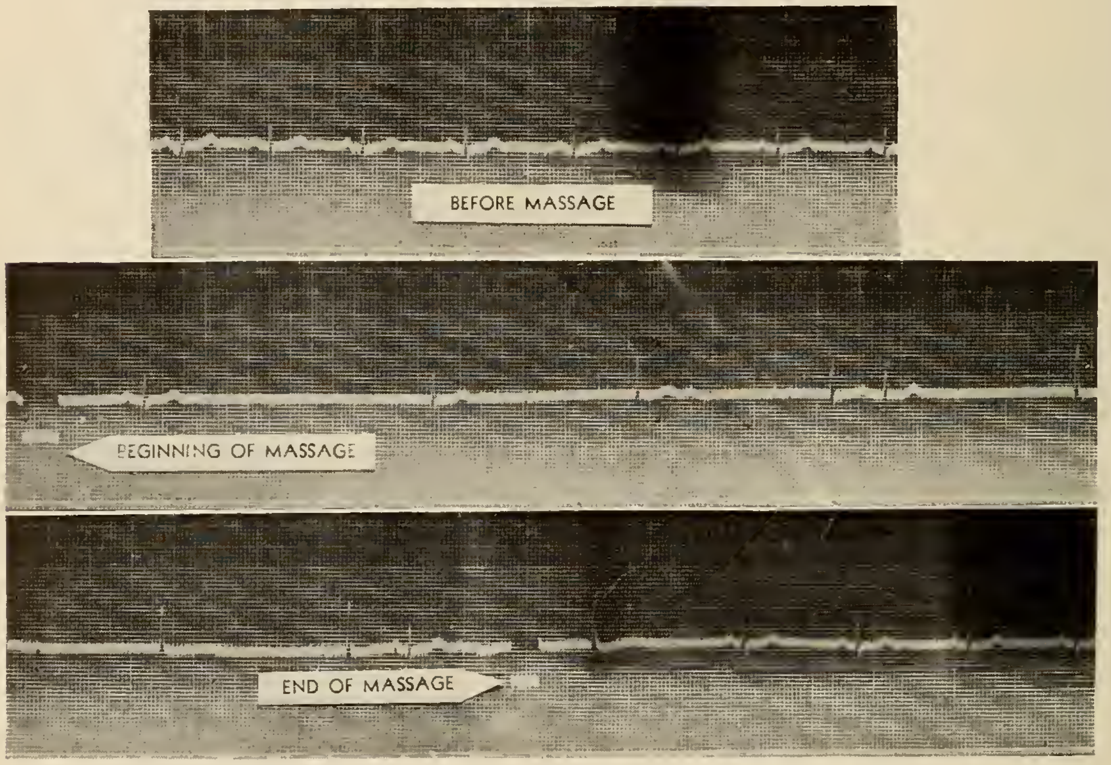

Fig. 2. Case No. 2. Electrocardiogram taken during massage of right carotid sinus.

No. 2. F. L. S., a white male, was admitted to the Veterans Administration Facility, Augusta, Georgia on January 6, 1939. The family and personal histories were essentially negative. His present illness started about ten years ago, at which time the patient began to show mental aberrations. He developed many visual and auditory hallucinations which eventually required treatment in hospitals for mental diseases. During previous hospitalizations he had been given diagnoses of general arteriosclerosis, psychosis with cerebral arteriosclerosis, and arterial hypertension.

Physical examination. The patient was a well-developed, fairly well-nourished, white male, age 61 years. The head and neck were essentially negative. The heart was not enlarged, there were no murmurs. Blood pressure was 140/80; pulse rate ranged from 64 to 72 per minute. The walls of the peripheral vessels were moderately thickened. Lung fields were clear. Abdominal examination was negative except for slight tenderness over Mayo-Robson's point. The genitalia, rectum and extremities were negative. Neurological examination was essentially negative, except that the deep reflexes were slightly hyperactive. Mental examination revealed poor memory, poor insight and defective judgment, as well as many auditory and visual hallucinations and delusions of grandeur.

Special investigation of the carotid sinus. Attention was directed to the carotid sinus because the patient placed his finger directly over the right carotid sinus and said, "My tonsil hurts here," and also because there was a history of dizzy spells, and, as the patient stated, a feeling that his head was swelling. Massage of the left carotid sinus on one occasion caused the blood pressure to fall from 124/86 to 114/84, but the pulse rate remained constant at 72 per minute. On another occasion stimulation of the left carotid sinus caused the blood pressure to change from 126/88 to 134/90, and the pulse again remained stationary at 72 per minute. Massage of the right carotid sinus, however, produced dramatic effects. The blood pressure dropped from 138/96 to 86/66, and the pulse rate fell from 76 to 46 per minute. The patient complained of dizziness and the feeling that he was going to sleep. When massage on the right side was repeated a few minutes later, the blood pressure dropped from 116/84 to 74/50, the pulse rate diminished from 72 to 48 per minute, and the patient complained of dizziness. No actual syncope followed stimulation of the sinus. As in Case No. 1, the patient evinced more tenderness on palpation of the hypersensitive sinus than on pressure over the normal sinus. Figure 2 illustrates the profound slowing of the pulse, with suppression of sinu-auricular activity and idioventricular rhythm upon stimulation of the right carotid sinus.

SUMMARY

1. The hyperactive carotid sinus reflex is a clinical entity described comparatively recently, and is characterized by a definite symptomatology which can be artificially reproduced.

2. The etiology, symptomatology and treatment have been discussed.

3. The method of eliciting the reflex by massage of the carotid sinus has been described.

4. Two cases representative of hyperactive carotid sinus reflex are reported. Case No. 1 is principally of the cerebral type, as the pulse and blood pressure changes are not sufficient to account for the syncope. Case No. 2 is characteristic of the vagal-depressor type, indicated by depression of the blood pressure and pulse without accompanying syncope.

5. It is necessary to emphasize the importance of bearing this condition in mind when dealing with cases of syncope or convulsions of unknown origin.

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THE MODERN TREATMENT OF SCOLIOSIS*†

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Scoliosis, according to most authorities, is lateral curvature of the spine. Lovett (1) defines the deformity as a condition in which any series of vertebral spinous processes shows a constant deviation from the median line of the body, a deviation always accompanied by twisting or rotation.

CLINICAL FEATURES

The outstanding feature of this deformity is the disparity between the physical signs and the symptoms. It is not uncommon for a patient to present himself for treatment for the first time with a severe curvature which is hopelessly beyond the possibility of complete correction. He may have no back pain and no undue fatigue and be in good general health. Only patients with very severe angulated and rotated curves show any degree of pain or undue fatigue. In later years scoliotic spines are more likely to develop painful osteoarthritic changes earlier than do normal spines.

The best form of treatment is prophylaxis but since the causes are idiopathic in 60 per cent of the cases, the result of poliomyelitis in 30 per cent, congenital in 5 per cent and about 5 per cent in other conditions, it is obvious that one is as yet unable to prevent congenital deformities, poliomyelitis or idiopathic conditions. Early recognition is therefore of the utmost importance in the successful treatment of this deformity. Since scoliosis is a condition occurring during the growth period, it is important to scrutinize the unclothed back of every child one is called upon to examine. This should be done whether a deformity is suspected or not and the diagnosis can be made by inspection alone. The severe curves are obvious and require no special mention. It is in the early recognition of the mild cases which offer the possibility of complete correction that most good can be done.

The normal spine should support the head and trunk and center them directly over the pelvis. Furthermore, the normal spine corrects any temporary deviation of the trunk from the midline without any volition on the part of the individual. With the spine thus centered over the pelvis, a perpendicular line dropped from the tip of the spinous process of the first cervical vertebra should intersect the remaining spinous processes and bisect the sacrum. In mild curvatures this is at times difficult to observe. One must keep in mind that another characteristic of the normal back is its perfect symmetry. In cases of a loss of symmetry to the back scoliosis should be strongly suspected. One shoulder may be higher than the other, or a scapula may be elevated or more prominent owing to the rotatory deformity of the thoracic cage. In lumbar or thoracic curves that have been accompanied by a lateral trunk shift one hip may be more prominent than the other. Roentgenographic studies always show the actual curvature to be much greater than would be suspected from a clinical examination of the patient. Forward bending will bring out any slight asymmetry of the back that may not be apparent when the patient is standing or sitting.

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† Read before the Postgraduate Section on Pediatrics, American College of Physicians, at Harriet-Lane-Home for Invalid Children, March 16, 1939.

PROGNOSIS

A careful study of the prognosis of untreated scoliosis was made and reported in 1937 by Risser and Ferguson (2). They found that regardless of the cause of the curvature the deformity increased until growth had ceased and that after cessation of growth there was no further increase in the deformity. Lesions which usually caused scoliosis failed to produce the deformity if the onset occurred after growth had ceased. They also observed that boys averaged seventeen years and girls sixteen years as the age of growth cessation. It is obvious from these facts that any form of treatment used after the growth period has passed is useless as a means of preventing an increase in the deformity.

If these facts are kept in mind it should be possible to estimate the appearance of the back at the age of growth cessation from the probable amount of expected growth of the spine.

In the modern treatment of scoliosis the three cardinal principles laid down by Hoffa (3) should be applied. They are mobilization, correction and stabilization. The first two principles were successfully applied many years ago, but only since the development of the spinal fusion operation by Hibbs (4) and others and its application to curvatures has maintenance of the previously gained correction been possible. This operation was employed after maximum correction in a hinged jacket devised by Risser (5) and has since been improved upon by others.

TREATMENT

A suitable candidate for treatment should be chosen. The patient is placed upon a frame and a jacket applied enclosing the head, neck,

Fig. 1

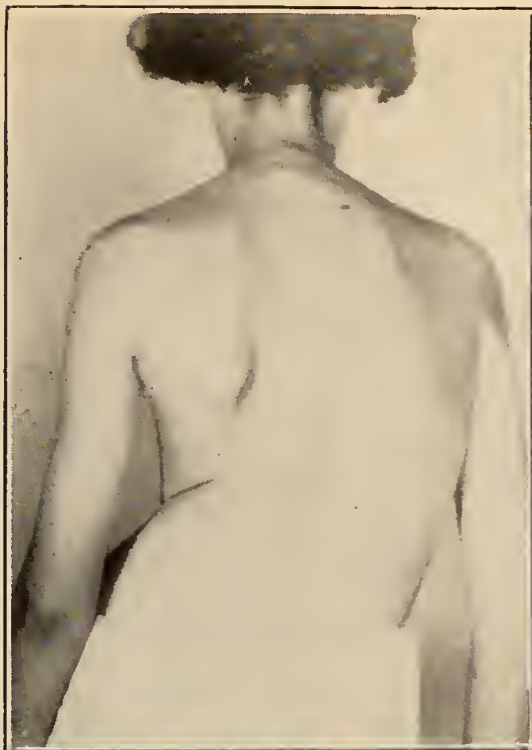
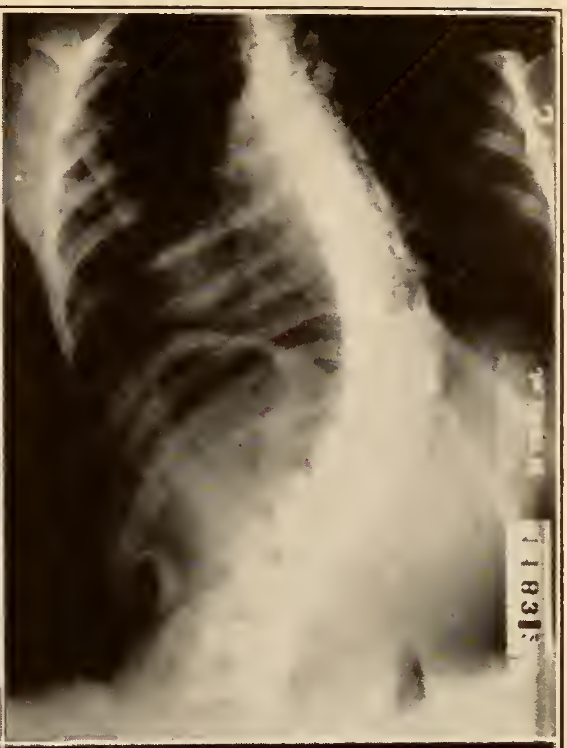


Fig. 2



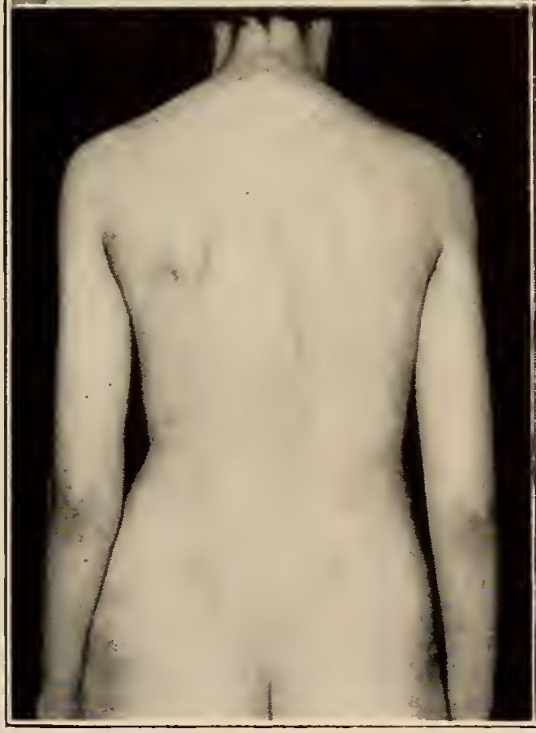


Fig. 3

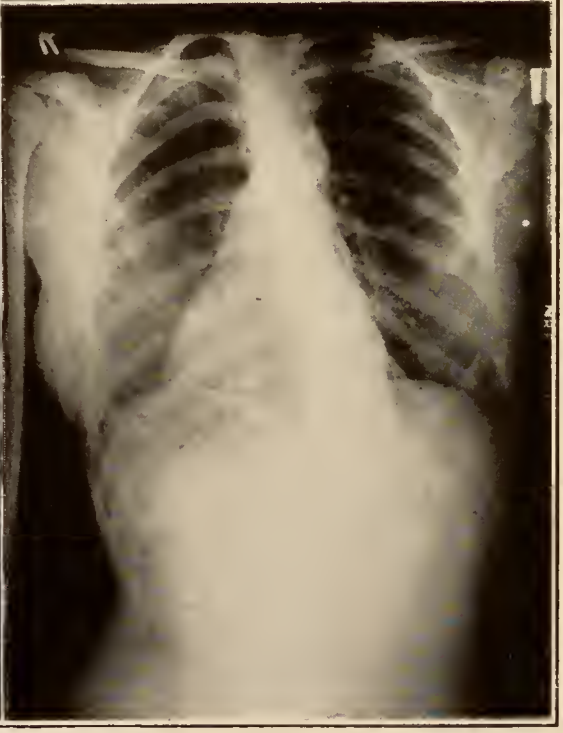


Fig. 4

Fig. 1. Preoperative photograph.

Fig. 2. Preoperative roentgenograph.

Fig. 3. Postoperative photograph.

Fig. 4. Postoperative roentgenograph. Patient has since married and gone through a normal pregnancy with no complications.

trunk, pelvis and the thigh on the convex side of the curve under correction. Incorporated in the plaster of this jacket are two hinges, one in front and one in the back, with their joints level with the apex of the primary curve. When the jacket has become sufficiently dry, a segment of plaster is taken from the convex side of the jacket and a saw cut is then made between the hinge joints. A turn-buckle is inserted and the jacket opened up a little each day, or as much as the patient can easily tolerate with a slight degree of discomfort. When the curve has been corrected as much as the patient can bear without extreme discomfort, or when the curve has been completely corrected, the back of the jacket is reinforced with splints. This is done in order to maintain stability of the jacket after the posterior hinge has been removed and fenestration done prior to operation. With a jacket of this type maximum correction can be obtained in from two to four weeks after its application. During the correction process one must watch for signs of undue pressure and for pull on the trunks of the brachial plexus on the concave side of the curve under correction. High dorsal curves must be corrected very carefully because of this complication.

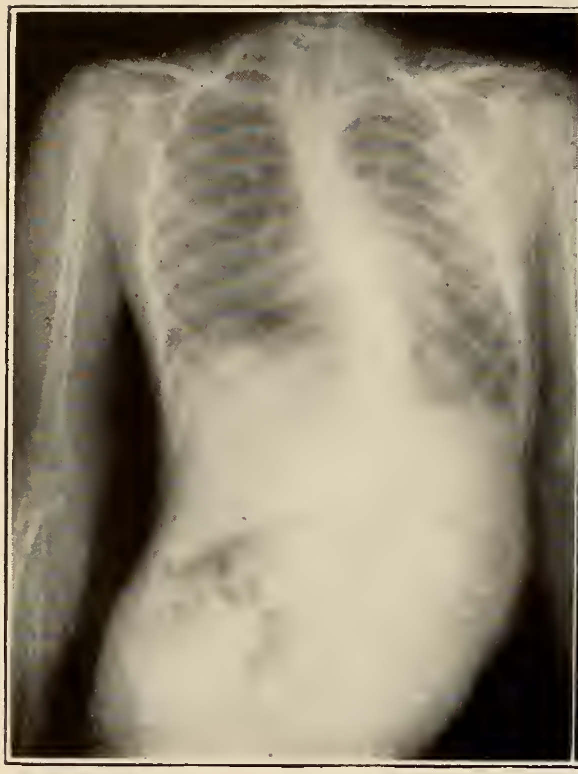


Fig. 5

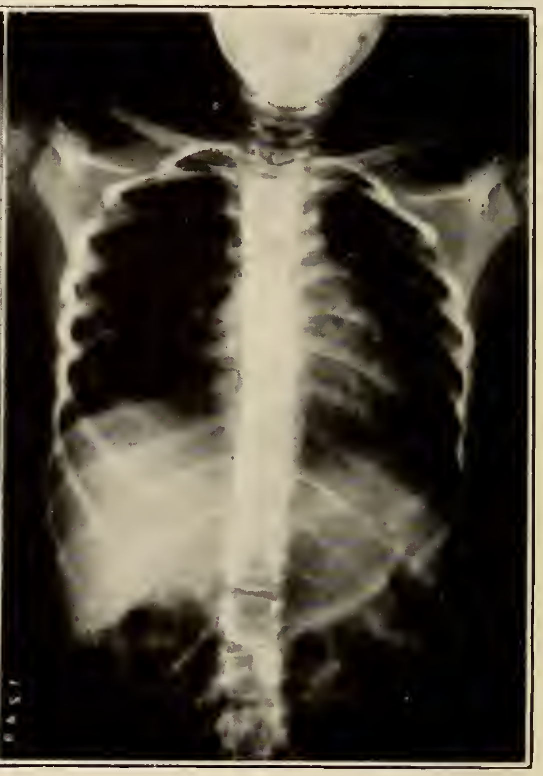


Fig. 6

Fig. 5. Patient aged 6. Preoperative roentgenograph of patient sitting. Scoliosis caused by poliomyelitis.

Fig. 6. Roentgenograph of patient in sitting position, one year after operation.

After correction of the curve and fenestration of the jacket the area of fusion should be selected. In cases where correction has been complete the limits of the fusion area are the ends of the primary curve. Where only partial correction has been possible and there is good flexibility to the remaining portion of the spine, the fusion area includes two vertebrae at the ends of the curve whose surfaces are parallel to each other and at right angles to a line joining the center points of their bodies. When there is not sufficient flexibility in the compensatory curve to correct completely, the fusion should extend to a vertebra with sufficient tilt to let the remaining secondary curve balance the residual primary curve.

At operation an incision is made over the tips of the spinous processes of the vertebrae to be fused. By clean subperiosteal dissection the overlying tissues are reflected as far laterally as possible to expose the lateral articulations. These are denuded of their cartilage and the interlaminar ligaments, together with the interspinous ligaments removed by sharp curettage. Chips from adjacent laminae are turned over the interlaminar spaces. Particular attention is paid to the apex of the curve where the stresses are the greatest. When the fusion is done in the lumbar spine it is reinforced with small chips taken from the tibia.

Where more than five joints are to be fused the operation is carried out in two stages. It is extremely important to include the correct number of vertebrae for an error of one or two vertebrae at either end may vitiate the expected result and leave the patient little better off than if he were untreated.

Twelve weeks after the operative work has been completed, with a three-week interval between stages, the jacket is removed and the patient allowed to walk in a curved tubular jacket. This is worn for twelve weeks and then changed to another jacket which is worn for twelve additional weeks, making a total postoperative immobilization time of thirty-six weeks. After the last jacket has been removed the patient is allowed to resume his normal activities without wearing any support.

From a study of patients treated by this method the problems of scoliosis approached from this point of view when compared with regard to prognosis in untreated cases become clearer. Because of the lack of maintenance of correction in the growing patient, treatment by correction alone proved disappointing to any one who has followed his patients for any length of time. The effectiveness of any form of treatment must be judged by its ability to maintain correction in growing children.

SUMMARY

From careful follow-up studies on growing operative patients the following conclusions are drawn:

1. If the fusion is solid and adequate in extent there should be no increase in the curvature even in the presence of growth.

2. If there is a loss of correction the most probable cause is a pseudarthrosis or a break in the continuity of the fusion area. This should be repaired as quickly as possible before the previously gained correction has been lost.

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LIPOID STORAGE DISEASE*†

A CASE REPORT WITH AUTOPSY

IRVING FREEMAN, B.S., M.D. AND GEORGE F. SCHMITT, JR., M.D.

There is a group of diseases characterized by a disturbance of lipoid metabolism in which lipoids accumulate in the cells of the reticulo-endothelial apparatus and give them a characteristic large pale vacuolated appearance. For purposes of classification Rowland (1) has divided the group into primary and secondary generalized forms and primary and secondary localized forms.

The primary generalized forms are further divided into:

1. Idiopathic xanthomatosis, characterized by an abnormal deposit of lipoids normally found in the body fluids and tissues.
    - *a.* Acute—Niemann-Pick disease.
    - *b.* Chronic—Hand-Schüller-Christian syndrome.
2. Idiopathic xanthomatosis, characterized by deposits of a complex lipid, kerasin, not found in the normal body fluids and tissues; Gaucher's disease.

The secondary generalized forms include the xanthomatoses associated with diabetic lipemia, certain liver affections with jaundice, chronic glomerulonephritis, pregnancy, menopause, etc.

The localized forms may occur primarily or secondarily as a part of diseases associated with lipemia. These forms include the xanthomata of tendons and tendon sheaths, of the intima of blood vessels, of the skin, and scattered deposits in the spleen and liver.

More recently Thannhauser and Magendantz (2) have offered a new classification of xanthomatous diseases. They divide the group into a primary form with and without elevated blood cholesterol, a secondary form occurring with lipemia, and a form in which localized xanthoma cell formation occurs in true tumors.

The primary xanthomatoses are rare. There is only one case so classified in the records of the University Hospital. Recently a case falling into this group has been studied rather completely. In order to show its relationship to the better recognized clinical forms, brief descriptions of the latter are given as follows:

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Acute idiopathic xanthomatosis or Niemann-Pick's disease occurs chiefly in Jewish female infants and is frequently familial. It has a rapid course characterized by splenohepatomegaly, digestive disturbances, retarded mental and physical development, yellowish-brown skin deposits, moderate lymphadenopathy, hyperlipoidemia, slight secondary anemia, and leukocytosis. The lipoid lecithin, a phosphatide, is found in the cells of the reticulo-endothelial apparatus throughout the body. The spleen is greatly enlarged, red and firm. The malpighian bodies are not discernible. Characteristic large foam cells are found chiefly in the splenic pulp, and to a lesser extent in the liver, lymph nodes, lungs and kidneys.

Chronic idiopathic xanthomatosis, or the Hand-Schüller-Christian syndrome, has a more chronic course characterized by splenohepatomegaly, dwarfism, exophthalmos, diabetes insipidus, loose teeth, swollen gums, and yellowish-brown deposits in the skin and mucous membranes. Here the lipoid cholesterol is found in various parts of the body, more particularly in the membranous bones. The deposits near the apex of the orbit produce exophthalmos and diabetes insipidus as a result of pressure. The bones of the skull and pelvis have characteristic defects with ragged borders. The reticulo-endothelial cells in these areas contain foamlike cytoplasm in which the cholesterol is deposited. These same cells are present in the lymph nodes, spleen, liver and bone marrow.

Gaucher's disease is congenital and familial, involves females in the ratio of two to one, and is more common among Hebrews. The condition usually commences in childhood and is quite chronic. It is characterized clinically by marked splenomegaly, moderate hepatomegaly, emaciation, gross skeletal defects, cuneiform thickenings of the conjunctivae, anemia, and leukopenia. The arms and legs become thin in contrast to the enlarged abdomen. Retarded mental and physical development is common. The characteristic cell, found chiefly in the reticulo-endothelial apparatus of the liver and spleen, is large, pale, round to polyhedral in shape, and has an eccentric nucleus. The nuclei are frequently multiple. The cytoplasm contains the lipoid kerasin, a glycolipid or cerebroside not normally found in the body, and when stained with Mallory's aniline blue shows many blue fibrils.

Common, therefore, to all of the primary xanthomatoses is a hyperplasia of reticulo-endothelial cells with phagocytosis or storage of lipoids in the cells. Because of the variable character of the lipoids there are certain differences in staining reactions, which the following table from Krumbhaar and Custer (3) summarizes.

CRITERIA	GAUCHER'S	NIEMANN PICK'S	HAND-SCHÜLLER-CHRISTIAN'S
Shape of cell	Round, polyhedral, spindle	Round	Round
Lipoid reaction	Negative	Positive	Positive
Sudan III	Faint greyish-yellow	Orange	Orange
Nile blue	Diffuse light blue	Lavender	Lavender
Mallory's aniline blue	Blue parallel cytoplasmic striations	Light blue, no striations	Light blue, no striations
Turnbull's blue	Positive for iron	Negative	Variably positive
Weigert's iron hematoxylin	Pale bluish-grey	Bluish-black	Bluish-black

The case recently studied at the University Hospital presented many clinical features found in Gaucher's disease. Pathologically, however, by differential staining certain dissimilarities were apparent.

CASE REPORT

History. The patient was a 16 year old colored female admitted to the medical service of the University Hospital on November 26, 1937 because of convulsions during the previous night. For the preceding two weeks she had complained of cough, precordial pain, palpitation, and progressive weakness.

Her past history was interesting in that she had had repeated epistaxis since the age of three. She had measles, pertussis and chickenpox when a child. Physically she developed rather slowly and never attained adult size or developed secondary sex characteristics. An enlarged abdomen was noted at the age of twelve or thirteen. This enlargement had steadily progressed.

The family history was negative. No relative, immediate or distant, had a similar disease.

Physical examination. The physical examination revealed a stuporous state. The respirations were rapid and noisy. The temperature was 99.2°F., pulse rate 120, and respiratory rate 30. The conjunctivae and nail beds were somewhat pale. The state of development was distinctly below the given age. There were no secondary sex characteristics. The body length was 130 cm. The distance from the soles of the feet to the symphysis pubis was 65 cm. There was no generalized lymph node enlargement. Slight edema of the feet and shins and rather marked edema of the eyelids were noted.

The head was rather small, with prominent frontal bosses. The pupils were slightly dilated and the fundi showed no abnormalities. Thick, yellow-brown crusts filled the nares and there were impetiginous lesions about the nose and upper lip. There were many carious teeth, with an active gingivitis. The breath had a foul odor. Purulent material was present in the right ear canal. The mastoid areas were not tender. The isthmus and right lobe of the thyroid were palpable.

There was an outward flaring of both costal margins. The ribs were prominent and were drawn in along Harrison's groove during inspiration. There were numerous moist and musical rales over the entire lower half of the chest. By percussion the left cardiac border was 8 cm. from the midsternal line in the fifth interspace. The right cardiac border was 3 cm. from the midsternal line in the fourth interspace.

The transverse diameter of the chest was 21 cm. The sounds were soft, rapid and regular, with a protodiastolic gallop. The blood pressure was 170/110.

The abdomen was enlarged, particularly in the upper half. The liver was large, firm, and had a regular edge with a deep notch in the midline. Nothing was felt in the gallbladder region. The spleen formed a prominence in the left flank and was firmer than the liver. A large area of splenic dullness extended above the costal margin. There was little descent of the liver or spleen on deep breathing. The lower abdomen showed no evidence of free fluid.

The external genitalia appeared infantile and showed a scant purulent exudate with a mild inflammation of the mucous membrane of the vagina.

The extremities showed slight edema of the feet and legs, and old scars along the tibiae. The deep reflexes could not be obtained.

Laboratory data. The urine was persistently acid and had a specific gravity of 1.010 to 1.018. It contained albumin (4 plus), numerous white blood cells, red blood cells, and hyaline and granular casts. The phenolsulphonphthalein excretion was zero at the end of two hours.

The blood picture was as follows: red blood cells 3,270,000; hemoglobin 60%, Sahli; white blood cells 7,950; neutrophilic leukocytes-filamentous 76%, nonfilamentous 5%; lymphocytes 15%; monocytes 3% and myelocytes 1%. No sicklying of red blood cells was observed. Bleeding time was 4½ minutes and clotting time 12 minutes. Reticulocytes made up 0.6% of the red cells. The volume index was 0.8, the fragility test was normal and the sedimentation rate (Wintrobe) was 26 mm. in one hour. There were 382,000 platelets per cu. mm.

On admission, the blood chemical examination was reported as follows: nonprotein nitrogen 183 mg.%; creatinine 7.4 mg.%; uric acid 10.5 mg.%; sugar 116 mg.%; carbon dioxide combining power 24 vol.%; cholesterol 384 mg.%; van den Bergh—delayed, direct reaction, trace; calcium 9.3 mg.%; phosphorus 9.9 mg.%; albumin-globulin ratio 2.06/3.1.

The stool examination was negative.

Intradermal tuberculin test (0.005 mg.) was negative.

The Congo red test for the presence of amyloid revealed 37.5% absorption of the dye at the end of one hour, a borderline result.

Escherichia coli communior was cultured from a catheterized urine specimen, but a later culture yielded a streptococcus nonhemolyticus. The blood culture was persistently negative. A smear from the gums revealed an abundance of Fusiformis (dentium) and Borrelia vincenti organisms, and a Corynebacterium flavidum was grown from the ear discharge.

The bromsulphalein liver function test showed 5% of the dye to be retained after one-half hour and 3% after one hour, evidence of significant liver damage.

Glycogen storage disease was ruled out by the following: several fasting urine specimens revealed no trace of acetone; a number of fasting blood sugar determinations were within the normal range (116 to 119 mg.%); a significant rise in fasting blood sugar (118 to 129 mg.%) was obtained with the hypodermic administration of 6 minims of 1:1000 adrenalin.

X-ray examination of the left hand and wrist, the lower lumbar and sacral regions of the spine, the upper ends of both femurs and the skull showed nothing pathological. That of the chest showed an extensive infiltration of both lungs, splotchy in character on the right side, with an area of consolidation in the central portion of the lung on the left side. The paranasal sinuses showed clouding of the left antrum and ethmoid cells and the sphenoid. A film of the abdomen showed an enlarged liver and spleen, with the kidneys indistinctly visualized but apparently not grossly enlarged.

The electrocardiogram revealed T_1 inversion, late T_4 inversion, and left axis deviation.

Course in hospital. The patient was given a liquid diet. Fluids were forced orally and by clyses; alkalinizing solutions were given by the same routes. Rapid digitalization was instituted. The draining ear was irrigated with warm boric acid solution. Ammoniated mercury ointment (10%) was applied to the impetiginous lesions on the face. Within several days left blepharitis developed, which responded to application of yellow oxide of mercury ointment.

The patient's general condition improved slowly during the first week. She became more alert, her respirations were less labored, and the pulse rate dropped to 72. The edema, however, increased and evidence of free fluid in the abdomen was noted.

From the time of admission until December 16, 1937 the rectal temperature at no time exceeded 100.2°F., the pulse never rose above 110, and the respirations remained in the neighborhood of 30. The blood pressure ranged from 120 to 190 systolic and 80 to 126 diastolic.

On December 13, 1937 the blood chemical examination showed nonprotein nitrogen 206 mg.%, creatinine 8.1 mg.%, and carbon dioxide combining power 28 vol.%. The urinalysis and blood picture did not change.

At 5:00 A.M. on December 16, 1937 the patient developed a sharp pain in the left lower chest and upper abdomen which was worse on coughing and deep breathing. No friction rub could be heard, but the breath sounds at the left base were bronchovesicular to bronchial and were accompanied by numerous crackling rales. By 4:00 P.M. the temperature rose to 103.6°F., the pulse to 120, and the respirations to 40. Respirations ceased at 8:00 P.M. on the same day.

Pathology. The autopsy was performed eleven and one-half hours after death. The body was that of a poorly nourished, poorly developed, colored female child, measuring 130 cm. in length. Small excoriated areas were seen about the nose and upper lip. There were no secondary sex characteristics. Moderate pitting edema of the legs and ankles was present.

The peritoneal cavity contained 700 cc. of straw-colored fluid having a specific gravity of 1.016. The peritoneal surfaces were smooth. The liver and spleen were much enlarged.

The pericardial cavity contained 100 cc. of straw-colored fluid. The myocardium was pale and flabby. The valves appeared normal. In the intima of the aorta there were scattered, yellowish, linear atheromatous plaques.

The left lung weighed 650 gms., the right 610 gms. There were scattered areas of consolidation in the left lower lobe.

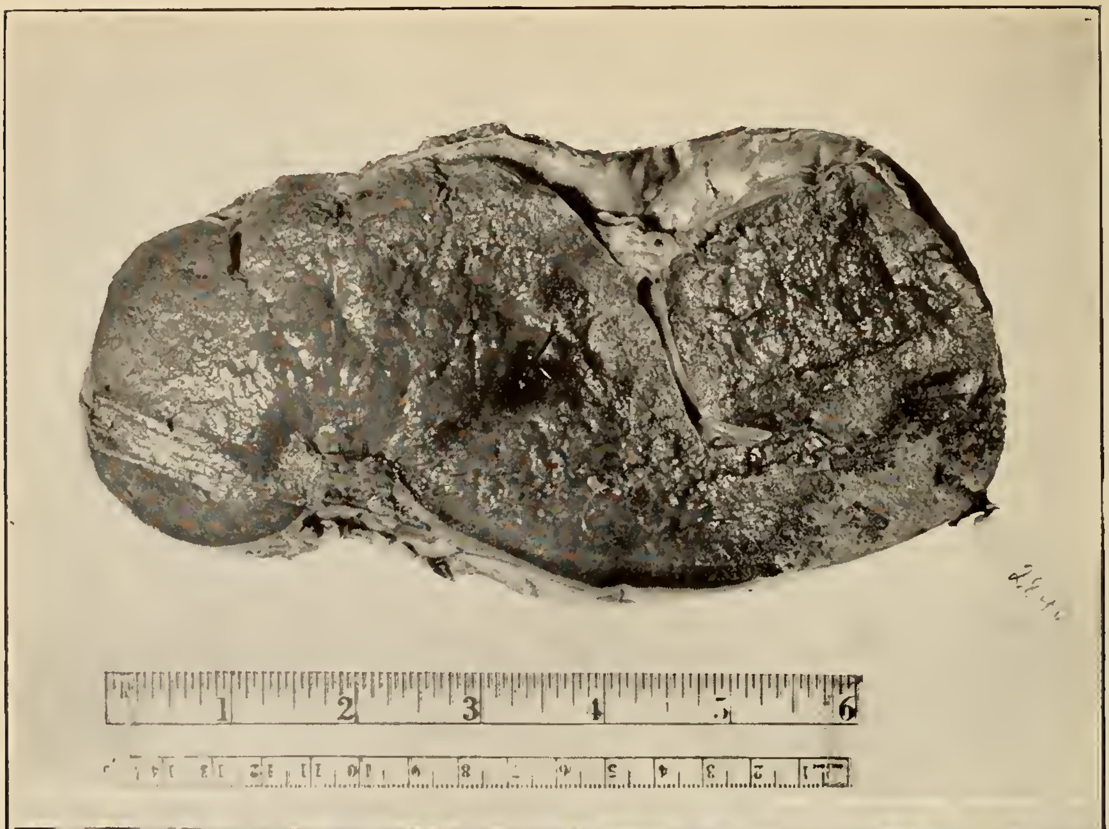
The spleen weighed 1,500 gms. and measured 22 by 13 by 7.5 cm. The capsule averaged 2 to 3 mm. in thickness and simulated white cake icing. The pulp was red and had a puttylike consistency. The malpighian bodies were not visible. In the lower pole there was a sharply defined, firm, yellowish-white area measuring 3 by 1.5 cm., which was surrounded by a hemorrhagic zone.

The liver weighed 2,650 gms. and measured 28 by 21 by 8 cm. The parenchyma had a pale yellow color. Scattered throughout were numerous petechial hemorrhages bearing no constant relationship to the central vein zones.

The left kidney weighed 75 gms., the right 125 gms. The pelves and ureters were not dilated. The capsules stripped with difficulty and left granular surfaces. In several of the columns of Bertin were noted yellow, granular, fatty-appearing deposits.

The other organs presented no gross lesions.

FIG. 1



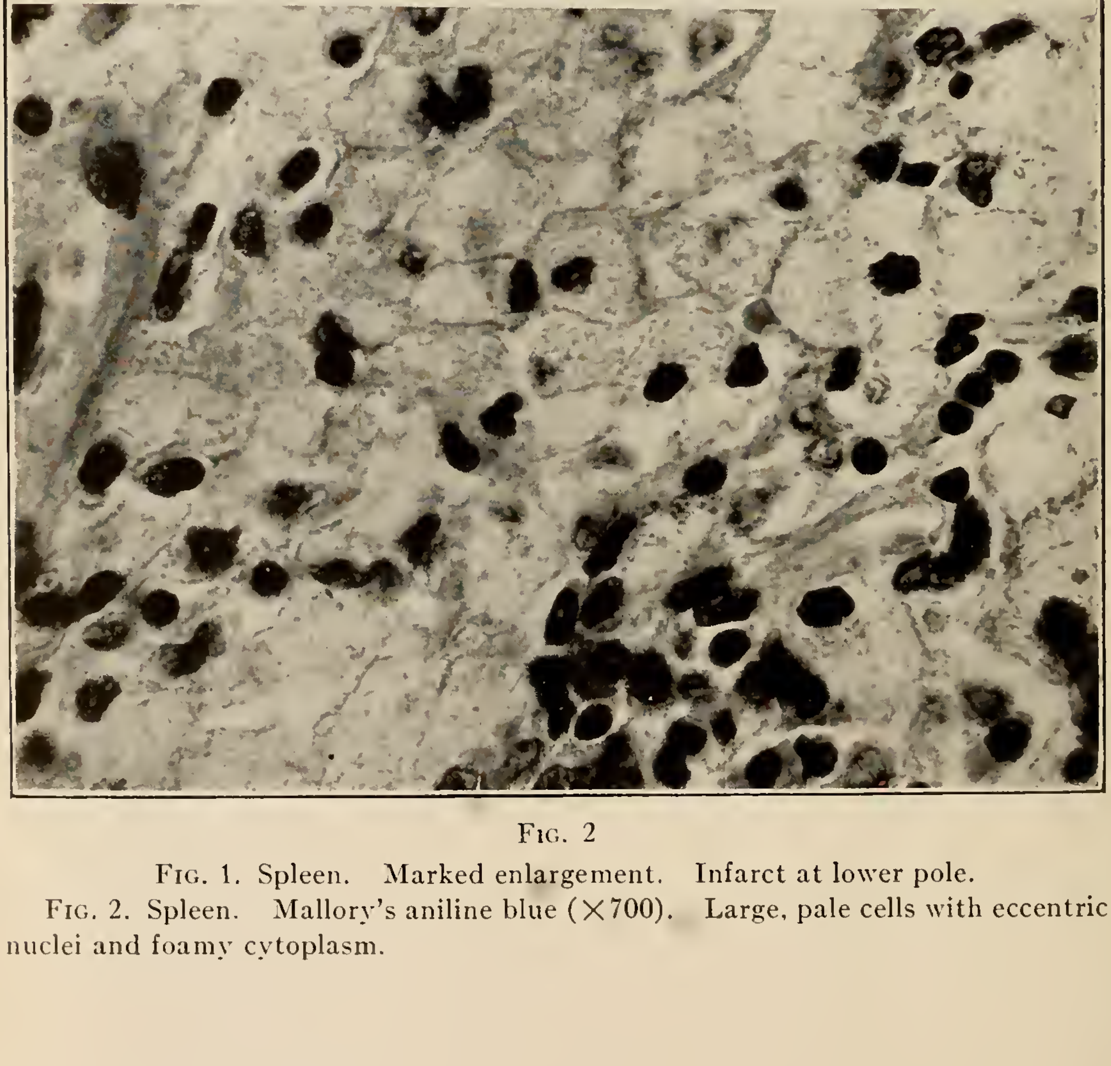


FIG. 2

FIG. 1. Spleen. Marked enlargement. Infarct at lower pole.

FIG. 2. Spleen. Mallory's aniline blue (×700). Large, pale cells with eccentric nuclei and foamy cytoplasm.

Microscopic. Aorta: In the intima were many widely separated large oval cells, each having pale, granular cytoplasm and a single, deeply stained, eccentric nucleus.

Lung: Most of the alveoli were filled with an exudate containing granulocytes and large ovoid cells with small eccentric nuclei. The cytoplasm was pale and granular, and contained brown pigment. The exudate was also noted in the smaller bronchi. Sections stained with Turnbull's blue showed that the brown pigment in the cytoplasm was iron.

Spleen: The malpighian bodies were not discernible. The pulp contained great numbers of large oval cells, with pale fibrillary or granular cytoplasm and small deeply stained eccentric nuclei. The cells had a mean diameter of 50 microns and were occasionally multinucleated. Sharply demarcated from the remainder of the tissue was a necrotic area surrounded by a moderately thick zone of hemorrhage. On the capsule over this area was a fibrinous exudate.

Stained with Mallory's aniline blue, the cytoplasm of the large cells was shown to consist of numerous fibrillae, which did not run parallel but rather interlaced; with sudan III the color varied from a grey-orange to a red-orange; with Turnbull's blue and Prussian blue, no iron was demonstrable; with Nile blue sulphate, the cytoplasm stained a diffuse light blue; with Weigert's iron hematoxylin, the cytoplasm stained a greyish-brown.

Liver: About the triads were fine scarred areas in which were evident large, pale, oval cells with eccentric nuclei. The cytoplasm had essentially the same staining reactions as that in the cells of the spleen.

Kidney: The sections showed glomerular scarring and focal interstitial fibrosis. The cells of the convoluted tubules were swollen and granular, and in many instances had desquamated into the lumens of the tubules.

Bone marrow: The section showed hyperplasia with a few focal areas in which pale vacuolated cells with eccentric nuclei were noted.

Anatomical diagnosis. Hepatomegaly; splenomegaly; cirrhosis of the liver, portal; chronic glomerular nephritis; infarction of the spleen; perisplenitis; emaciation; lobular pneumonia, confluent, left lower lobe; hydroperitoneum; hydropericardium; subcutaneous edema, legs, thighs; atherosclerosis, aorta; absence of secondary sex characteristics; excoriations, nose, upper lip.

COMMENT

Clinically, this case presented many of the features of Gaucher's disease. The onset in childhood, the occurrence in a female, the chronic course, the splenomegaly and hepatomegaly, the thin arms and legs contrasted to an enlarged abdomen, the retarded physical development, the emaciation, and the anemia all fit in well with classical Gaucher's disease. The absence of a leukopenia may be explained by an accompanying bronchopneumonia. Cuneiform thickenings of the conjunctivae adjacent to the cornea and gross skeletal defects were absent. The hypercholesterolemia could have been the result of renal failure on a basis of chronic glomerulonephritis beginning in early childhood. This could account for the entire syndrome, which would then be classified under the secondary generalized xanthomatosis outlined by Rowland (1).

Pathologically, the case presented retarded physical development and hepatosplenomegaly. The characteristic cell found in the splenic pulp and in the portal connective tissue of the liver was a large, pale, oval cell with an eccentric nucleus and fibrillary cytoplasm. By differential staining the cytoplasm was found to contain a lipoid and cytoplasmic striations, and to give a light blue staining reaction with Nile blue sulphate, typical of Gaucher's disease.

With Weigert's iron hematoxylin the greyish-brown reaction was typical of none of the three diseases.

The absence of iron reaction with Turnbull's blue coincided more with the reactions in Niemann-Pick's disease and the Hand-Schüller-Christian syndrome. Bloem, Groen and Postma (4), however, hold to the opinion that this reaction is not constant.

The portal cirrhosis and infarction of the spleen could well be a part of Gaucher's disease. The chronic glomerulonephritis may have been a prime factor in the causation of the syndrome or may have occurred incidentally. The confluent lobular pneumonia, in all probability, was a terminal event in the uremia of chronic nephritis.

CONCLUSION

It may be stated, therefore, from an analysis of the available data that the case represents a lipoid metabolic disorder of an unclassifiable nature, but that it closely resembles Gaucher's disease.

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RENAL DAMAGE RESULTING FROM PYELITIS IN PREGNANCY*

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INTRODUCTION AND DEFINITIONS

Pyelitis, according to the urologists Keyes and Ferguson (24), means, clinically speaking, inflammation of the pelvis of the kidney, of its ureter and of the kidney parenchyma as well.

According to Traut (37) there is some confusion in terminology. He explains that, as the name indicates, pyelitis is an inflammatory disease of the pelvis of the kidney. However, when the renal pelvis is inflamed the ureter also is usually involved, hence it is more accurate to use the term pyelo-ureteritis instead of pyelitis. If the infection extends from the renal pelvis into the parenchyma of the kidney so as to involve the ducts and secretory elements of the kidney, one speaks of pyelonephritis.

The subject of this paper is one which is indeed relatively unexplored. Whereas ever since 1841 pyelitis has been known to be a frequent concomitant of pregnancy (33), it is only in the last few years that the resulting renal damage has been investigated. The *Index Medicus* was searched from 1918 to 1939 without finding a paper whose title mentioned renal damage resulting from pyelitis in pregnancy. Among the first studies reported on the after-effects of pyelitis in pregnancy was that published by E. Granville Crabtree in 1929 (6). This worker discussed the rate of recovery after pyelitis in pregnancy. As recently as 1932 Dodds (16) observed that although many investigations have been made on the etiological factors in pyelitis of pregnancy and although there is a vast amount of literature on the subject, there is but scanty information and very little literature on the late results of this condition.

This investigator remarks that the general impression among obstetricians seems to be that the condition is cured by delivery, or even before delivery, after the acute attack has subsided, when, as a rule, these patients are asymptomatic. Such patients are not infrequently discharged from hospitals as well patients. It will be seen that pyelitis is a very serious complication of pregnancy and is not to be dismissed as a minor incident.

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According to Traut (37), pyelitis has been one of the least understood of the obstetrical diseases. This should be true no longer for recently much light has been shed upon the physiology of the ureter in pregnancy and the etiology of pyelitis on the one hand, while on the other diagnostic methods and the treatment of the disease have been improved and an understanding of the sequelae has been reached. This new information has necessitated a complete revision of ideas concerning the significance of pyelitis in pregnancy so that one is now forced to consider it as one of the major complications. Many of the data which have brought about this change in point of view have been obtained by means of improved methods of urologic diagnosis. More important, however, has been the accumulated information which has resulted from cooperation between urologists and obstetricians in obstetrical hospitals both in this country and abroad.

INCIDENCE OF PYELITIS IN PREGNANCY

The incidence of pyelitis in pregnancy is marked by wide variations in figures reported in the literature. As Douglass (17) remarks, the incidence is usually greatly underestimated, there being several reasons for this. Many of the attacks are mild and have a tendency to subside spontaneously, so that they are either not reported or are undiagnosed. Only the individuals with more severe cases are admitted to hospitals and even in reports from these institutions are found discrepancies. Pyelitis, he concludes, probably occurs in varying degrees of severity in about 25 per cent of all pregnant women.

Crabtree, urologist at the Boston Lying-In Hospital, gives an incidence in 4,000 prenatal cases of 1 per cent (6).

Dodds, obstetrician at the University College Hospital (England), in a study of 7,512 cases from 1927 to 1930 reports an incidence of 1.1 per cent antenatal cases (16).

At the Southern Baptist Hospital, among 2,400 women admitted for delivery 2.4 per cent had pyelitis antepartum (39).

Traut (37) points out that pyelo-ureteritis as a complication of pregnancy is sufficiently important to classify it with chronic nephritis and cardiac disease. In 15,000 consecutive deliveries at the New York Lying-In Hospital, Traut found the following comparison: pyelitis 2.02 per cent, chronic nephritis 2.29 per cent, cardiac disease 3.66 per cent. He remarked that not only does pyelo-ureteritis rank with chronic nephritis and cardiac disease in frequency, but if neglected the sequelae may also cause it to rank in importance with these diseases.

Dugald Baird (1) reports an incidence of 16.3 per cent in 1,000 consecutive admissions to Glasgow Royal Maternity and Women's Hospital. In 1936 the same worker (2) reported an incidence of 15 per cent at this hospital. His figures are the only ones approaching the incidence which Douglass (17) assigns to this complication of pregnancy.

The incidence of postpartum pyelitis is reported variously at .4 per cent (9, 10), 1.6 per cent (16), and 1.5 per cent (39).

PATHOLOGY

Thorough knowledge of the pathology of pyelitis in pregnancy is essential for an understanding of the damage which follows. It will not be amiss to review briefly the more important findings.

As Eastman (18) remarks, bacteriuria is common in pregnancy and urinary stasis is the rule. It is, therefore, surprising that pyelitis is not more frequently a complication. Presently it will be seen that the urinary tract undergoes marked physiological changes in pregnancy and that these closely border upon and easily become diseased conditions.

Traut states that 80 per cent of pregnant women have some degree of hydro-ureter and hydronephrosis (38). In discussing the dilatation of the ureters in pregnancy, Keyes and Ferguson (24) report that the ureterectasis of pregnancy is a condition that many urologists seldom see, yet they should be familiar with its peculiarities for it provides the ideal opportunity to study the effects of slight back pressure upon the ureter, the beginnings of hydronephrosis, the hydrodynamics of ureteral retention of urine, and the influence of urinary stasis in occasioning infection of the urinary channels. The kidney, they continue, does not show the lesions of hydronephrosis except to a slight and temporary degree. The ureter below the pelvic brim is little if at all dilated; it is rather the abdominal portion of the ureter that dilates.

In exceptional cases one or both ureters are dilated to an enormous size and a true hydronephrosis may promptly follow. Even when greatly dilated the impairment of renal function is not sufficient to occasion nausea and vomiting above the ordinary. Symptoms are not to be expected unless the pool of residual urine becomes infected.

Hence, it will be noted from the above discussion that dilatation of the ureters occurs during pregnancy and that this change means urinary stasis with possible progression of the changes to hydroureter and hydronephrosis. Obviously, urinary stasis plus infection means ureteritis, pyelo-ureteritis or pyelonephritis.

The cause of ureteral dilatation in pregnancy should be briefly considered. Several investigators have studied this point and a number of explanations are offered. Crabtree (7) advocates that the pressure from a growing uterus can be the cause. Baird (1) says that intravesical edema with constriction of the ureters is a factor. Cumston (14) believes that torsion of the uterus with stretching of the left ureter and kinking of the right is the cause. Hofbauer (22) has demonstrated that thickening and hypertrophy of the juxtavesical portion of the ureter occurs and is the basis for ureteral dilatation. Hundley, et al. (23) have demonstrated a relaxation and atony of the ureter from a hormonal cause.

Most observers agree (and it has been shown roentgenologically) that this dilatation takes place sometimes as early as the sixth week, before the uterus is large enough to cause compression anywhere near the ureter and before torsion occurs. Also, that the hypertrophy of the muscular and fibrous elements and the great thickening of the ureteral sheath of Waldeyer which occurs in pregnancy does not occur in the presence of larger and harder pelvic masses such as fibroids, etc. The urinary tract, then, which is so closely associated with the genital tract in origin, responds to the increased hormonal output of pregnancy by softening, dilatation, loss of tone, and muscular and fibrous hypertrophy of the ureter, most marked above the pelvic brim. Hundley, et al. (23) explain that the pelvic portion dilates little if at all because of the striking hypertrophy of the sheath of Waldeyer, muscle fibers which are present only in the pelvic portion.

This physiological change, therefore, is the all-important predisposing cause of the pyelitis of pregnancy.

A word may be said concerning the avenues of infection. These are threefold: by the blood stream, by the lumen of the ureter, and by lymphatics. In a recent exhaustive study of this subject Baird (2) has reviewed the work done in connection with the mode of origin and it is evident that no unanimity of opinion exists. With regard to the causal organism, however, there is general agreement that the Bacillus coli is the responsible agent in over 90 per cent of the cases.

Many investigators feel that pyelitis of pregnancy is a recurrence of pyelitis of infancy. Koll (25), Kretschmer and Falls (27), Williams (41), Pugh (32), and Rose and Rollins (34) are among those who have studied this aspect and believe that the infection is present from childhood in an attenuated form. The importance of this view will be referred to later.

Once pyelitis has occurred, it may be from the start and throughout its course no more than a bacteriuria or a pyuria (24). In mild cases the inflammatory process is confined to the mucous membrane of the pelvis and calices. Together with hyperemia and edema there is an outpouring of leukocytes, many of which accumulate on the mucosa in the form of an exudate; these become the clumped pus cells of the urine. Traut and Kuder (36) have emphasized with particular force the grave pathologic changes which ensue in prolonged cases. These are: (1) Involvement of the submucosa and muscularis with subsequent scar tissue formation, impairment of the blood supply and mobility of the ureters, which never completely involute after pregnancy terminates and so become the "fixed ureters of pregnancy". The woman is left with a urinary tract which harbors large amounts of residual urine and possesses little or no peristaltic activitity. Such a patient is extremely difficult to free of infection and she is particularly prone to a renewal of the acute phase in subsequent pregnancies. (2) The inflammation may extend beyond the renal pelvis and invade the parenchyma (pyelonephritis) when the danger of uremia is an immediate threat and cortical damage resulting from fibrosis and impaired kidney function is an ultimate possibility. (Traut directs particular attention to the value of examining the blood for its nonprotein nitrogen content in differentiating simple pyelitis from pyelonephritis.) (3) Finally, Traut described as the third serious process in the acute stage an extension of the inflammation from the pelvis of the kidney into the peripelvic tissues surrounding the large renal blood sinuses, with imminent danger of blood stream infections.

Sugar (35) points out that the infection will remain limited to the pelvis of the kidney as long as the urinary tract is unobstructed. However, as soon as there is an obstruction to the outflow of urine which produces a back pressure, the protective mechanism by which the spread of infection is kept from the kidney proper is broken down and the parenchyma of the kidney rapidly becomes infected from the pelvis by way of the perivascular lymphatic structures and by the direct passage of the organisms through the pelvic lining over the parenchyma.

RESULTANT RENAL DAMAGE

From the last few paragraphs some idea of the severe damage which may follow pyelitis in the pregnant woman is advanced. Before evaluating the damage which results from pregnancy pyelitis, one must know what the renal status was before pregnancy. Obviously, only adequate urological studies can determine this. It has been mentioned earlier in this paper that many observers believe that pyelitis of infancy and childhood, which was supposedly cured, is often reactivated by pregnancy. Wharton, Gray and Guild (40), discussing the late effects of acute pyelitis of childhood on the urinary tracts of a group of young women about ten years after their last attack of pyelitis, would appear to demonstrate persisting aftermaths of urinary abnormalities in some form or other in over 50 per cent of the cases.

In studying 300 pregnant women urologically, Crabtree, Prather and Prien (12, 13) found pre-existing renal abnormalities and pathology present in forty-four. These examples are cited to show the importance of judging aftermaths of pregnancy pyelitis in the light of previous pathology, a point which very few investigators have done as far as is determined from the available literature.

Bugbee (4) in 1918 showed some interesting foresight when he stated that renal function is such an important factor during pregnancy that it is right to look upon the pregnant woman in one sense as a urologic case. At that time he wondered about a very important and only recently developed aspect of urinary infection in pregnancy when he wrote "the stability of the relationship of bacteriuria to eclampsia and nephritis would be valuable". It will be seen that there is, indeed, a very real relationship. Other observers were beginning to realize that pregnancy pyelitis carried threats of serious renal damage, among them Davis, who wrote in 1918 (15) "the danger to the mother is the severe infection, permanent damage to one or both kidneys . . . ". Later, in 1921, Baughman (3) recognized that the termination of pregnancy was not the end of the pyelitis and he wrote: "even after delivery the pyelitis usually persists, infecting the kidney, reducing its function or crippling it entirely". In 1922 Hodges (21), after studying fifteen cases of pyelitis with pregnancy observed that the lesser and medium grades will entirely recover, but the more severe grades, with atony of the pelvic musculature, only partially recover. These early studies were the beginnings of much investigative work on this phase of the subject, work which necessitated close observation of patients even years after pregnancy.

FACTORS IN END RESULTS

One must remember that there are several factors which determine what the end results in the urinary tract will be. (1) Previous infection has already been mentioned. In a study based on 400 cases of diseases of the urinary tract associated with pregnancy, from 1919 to 1933, Crabtree and Prather (11) conclude that the history of cured pre-existing infection is not important in prognosis. The majority of infected patients entering on their first pregnancy will have febrile symptoms. When infection is present in the tract at the beginning of pregnancy, cure in pregnancy is an extremely rare occurrence.

Investigators in France, among them Lepoutre, President of the Obstetrical Society of Lille (28), state that pregnancy holds no special risk for a woman who has had the usual acute pyelitis outside of pregnancy and whose urine showed no pus or organisms after apparent cure. He states further that pregnancy on top of a chronic pyelitis, with pus and bacteria in the urine, does not cause a frequent risk and the acute recurrence is rarely grave. His paper, however, is lacking in figures supporting his conclusions.

(2) The length of time and the severity of infection and the amount of kidney involvement are important. For example, Hofbauer (22) found that no after-effects could be demonstrated when the signs and symptoms developed in the last ten days of pregnancy. Kretschmer (26) made pyelograms as early as the tenth day after labor in mild cases and showed that ureteral dilatations had almost completely disappeared. [Discussion of paper by Falls (27).] Crabtree (6) reports that the average time postpartum at which the less severe cases of pyelitis show recovery is three months. Severe cases of overdistention and infection of months' duration may have long convalescence or fail to recover entirely. Crabtree and Prather (9) feel that the effect of ureteral dilatation on the kidney during pregnancy is a consideration of supreme importance to the future life of the patient. They refer to Perineau's three grades of renal retention as sound conceptions by which to judge the problem of overdistention: *a.* retention of small amounts of urine in the renal pelvis where the pressure is not too severe, the fibers of the pelvis do not lose their tone under tension and there is a return to normal; *b.* in more pronounced dilatation the fibers are weaker but have not completely lost their tone and are capable of slower improvement and approach to normal; *c.* in extreme dilatation, the tissues are overstretched and replacement of normal structures by inelastic scar tissue fixes the pelvic walls and return to normal is impossible.

Crabtree (8) finds a periureteritis following pyelitis as the commonest cause of persistence of pregnancy changes into permanent deformity. This same investigator believes that pregnancy infections which fail to clear within four months postpartum do so because of four factors: debility of the patient, abnormal persistence of dilatations, presence of anomalies or gross pathology previous to pregnancy, and bladder damage with residual urine.

If pyelonephritis is long continued (meaning kidney involvement) it leads to a gradual patchy destruction of the kidney parenchyma. The parenchyma is thin, the capsule adherent, the markings are lost, the calices dilated in chronic infected hydronephrosis, and the functions low and fixed. Finally, the kidney is quite atrophied, its function lost (24).

(3) A third factor in the final outcome is the repetition of pregnancy. One may accept the obstetrical creed that, unless repeated more often than once in three years, the ureterectasis of pregnancy results in no permanent or progressive deterioration of urinary function (24). Rapid succession of pregnancies favors persistence of the infection and should be discouraged. The cure of urinary infection during pregnancy is not to be expected (6). It is almost certain that a patient who carries over an infection from a previous pregnancy into the next gestation will have an infected pregnancy. When infection clears between pregnancies, the cure is no guarantee of immunity in subsequent pregnancies as the recurrence rate is far above the normal incidence of infection in pregnancy [seven in a group of sixty-three reported by Crabtree and Prather, (11)]. Lepoutre (28) reports that after an attack of pyelitis with pregnancy which is not subsequently cured, the woman will certainly suffer with a pyelitis which may put her life in danger at the next pregnancy.

(4) A fourth factor is that of treatment. Pyelonephritis tends to render permanent the ischemic changes in the kidney and the atony and dilatation of the pelvis and ureter. Yet it is surprising how complete is the return of function after even the most acute pyelonephritis so long as the retention is relieved by catheterization before fixed hydronephrosis or pyonephrosis occurs (24). Hydro-ureter and hydronephrosis are likely to occur in those patients who are not discovered until too late or whose pyelitis does not receive adequate care, the mechanism being a fibrosis of ureter and pelvis (31, 38).

STATISTICS ON END RESULTS

Antenatal pyelitis. In regard to antenatal pyelitis, cure during pregnancy, as has been mentioned, is rare. Dodds (16) in a four-year study of cases reports only two cures before delivery. Crabtree (6) in a study covering seven years found only three cases.

In forty-eight antenatal cases Dodds found twenty-three; 47.9 per cent did not show any evidence of pyelitis or bacteriuria when examined two years after delivery. Of these, fifteen were cured within a year (16). Crabtree (6) made thorough pyelographic studies on twenty-six cases over a period of two years and found nineteen of the twenty-six were cured; ten cases were cured in two to eight months.

Baird (2) followed 208 cases of antenatal pyelitis over a period of one to five years and found that 32 per cent had recovered with sterile urine, and 16 per cent had apparently recovered but had infected urine. Of the remaining 52 per cent, all of whom complained of ill health or persistent renal pain, 21 per cent had sterile urine and 31 per cent had infected urine. In the 21 per cent group pyelography revealed persistent urinary tract dilatation and urea concentration tests indicated renal damage. In the 31 per cent group there was infection plus ureteral dilatation, with impaired urea concentration tests signifying parenchymatous involvement.

In Dodds antenatal group of forty-eight, twenty-five or 52.1 per cent had persistent infection. Eleven of twenty-five had bacteriuria alone and fourteen had chronic pyelitis. In this group of fourteen, three gave a history of previous pregnancy pyelitis, two were found to have renal calculi, and two had hydronephrosis.

In Crabtree's (6) group of twenty-six mentioned above, seven were not well at the end of two years. Of these, two had continued pregnancies at the rate of one a year; one had perinephritis with abnormal renal arteries; a fourth had a strictured ureter which existed before pregnancy, and a fifth had chronic pyelitis with bilaterally blunted calices.

Hofbauer (22) found seven out of fourteen antenatal pyelitis cases had a persistence of B. coli bacteriuria when examined some months later.

In Grieve's series of 100 cases he found forty were objectively cured and fifty-four subjectively cured. Of these fifty-four, forty were cured but still had infected urine (19).

Postpartum pyelitis. Postpartum pyelitis seems to be a less severe infection as well as a less common one. Dodds (16) had thirty-one cases, in which nineteen or 61.2 per cent were completely cured, fifteen within six months. Of the twelve not cured, three have chronic pyelitis and nine have bacteriuria alone.

Hofbauer (22) reports nine cases of postpartum pyelitis, all cured.

Cures and noncures. Complete cures in antenatal pyelitis, therefore, are reported as occurring in from 73 per cent (6) to 32 per cent (12). The renal damage resulting (aside from bacilluria) appeared permanent and is reported as frequent as 52.1 per cent by Dodds, 52 per cent by Baird, 50 per cent by Hofbauer, and 27 per cent by Crabtree. Compared with these figures the few reports on postpartum pyelitis appear to stamp it as much less severe. In considering end results attention should be drawn to the condition of the urinary tract prior to pregnancy, to determine whether an infection or anomaly is present, and what effect subsequent pregnancies may have. Dodds (16) feels that the risk of recurrence of infection in subsequent pregnancies depends on the persistence of the infection or of bacteriuria in the interval between pregnancies. Her report cites three cases of recurrence in fourteen patients who suffered from antenatal pyelitis. In these three there was a persistence of pyelitis from the previous pregnancy.

The urgent importance of adequate treatment and careful follow-up is made plain by these facts. Recurrence of pyelitis in a subsequent pregnancy is usually the result of failure to rid the patient of infection following the previous pregnancy (12, 13).

Pyelitis, renal damage and the toxemias of pregnancy. There is much interesting work being done at present on the relation of pyelitis to the toxemias of pregnancy, chronic nephritis and hypertension, aspects which at first glance seem far removed from so simple and so common a condition as pyelitis.

Baird (1) believes that disordered function of the urinary tract may be the underlying cause in the obscure toxemias of pregnancy. He feels that stasis without infection may be the mechanism and that it is likely to be overlooked. He reports that 9.9 per cent of his toxemic cases were probably caused by urinary infection. In a study of thirteen women who died of pyelitis of pregnancy he found that a constant feature was the presence of abscesses in the substance of the kidney. In autopsies on cases of subacute and chronic pyelitis, where death was from other causes, he found healed abscesses in the kidney substance, contracted kidney, glomerular fibrosis and vascular thickening (2).

In a series of important papers Peters, et al. (30), have brought forth evidence to show that pyelitis complicating pregnancy is a major etiological factor in the production of the toxemias. Studying 320 cases of toxemias at the New Haven Hospital they found that 41 or 13 per cent of these women had pyelitis. If the incidence of pyelitis is accepted at about 2 per cent, as most investigators believe to be correct, these figures would indicate that among the toxemic patients the incidence of the disease is six times its usual frequency. Almost every clinical syndrome described among the toxemias is illustrated by one or the other of these forty-one cases. Twenty-five had "frank" toxemias (they do not define this term), of which nine were typical eclampsia. Four had hypertension and edema. The remaining twelve were not seen during their initial toxemic pregnancies, but were diagnosed on the history they gave and their subsequent courses.

The outcome in these cases was as follows: death in acute stages of toxemias—7; chronic residua (hypertension, arterial sclerosis, chronic hydronephrosis or chronic pyelonephritis)—23. There were about 27 per cent who developed hypertension or edema or both before pregnancy was terminated. This would suggest that pregnant women who have pyelitis are more than twice as likely to develop toxemia as gravid women in general.

In the cases coming to autopsy, those who died in the acute stages presented, in addition to pyelonephritis, the tubular and glomerular changes which are considered characteristic of eclampsia. The authors (30) do not intend to give the impression that pyelitis is or even may be the direct or remote cause of all pregnancy toxemias. They believe it plays a most important predisposing role, along with certain vascular and renal diseases. In discussing the pitifully poor prognosis of those surviving in this series, they say death usually comes after a long and lingering illness in which hypertension and its sequelae are in evidence.

In reviewing the work of Peters, et al., Eastman (18) points out that the relationship between pyelitis in pregnancy and the toxemias is a moot question. For example, Baird states that he never has seen toxemia follow pyelitis in an otherwise healthy primigravida and believes that the predisposing factors in the two conditions are opposed. He suggests that in toxemic patients the increased output of posterior pituitary principle, which is believed by some to be a constant accompaniment of toxemia, stimulates ureteral peristalsis, corrects urinary stasis and so prevents the occurrence of pyelitis. This opinion of Baird's is not supported by any case figures. Eastman also quotes Dodds who, from a small series of thirty-seven cases of toxemia finds the urine sterile in thirty-five, and states that bacteriuria is not more frequent in toxemia than in normal pregnancy.

In discussing the work of Peters, et al., Herrick (42) states that pyelitis in pregnancy usually exists apart from any manifestations of so-called toxemia. If one labels as toxemias those cases that have hypertension as a dominant feature, it may well be debated whether pyelitis commonly contributes to that type of toxemia. It is now well known, Herrick continues, that about one-half of the non-nephritic toxemias, those marked by hypertension, have as an end result cardiovascular disease with hypertension. The role of urinary tract infection in the origin and course of this disease, with or without pregnancy, is a matter of debate. Infection, he feels, may accelerate the progress of some of the secondary degenerative changes in the cardiovascular tree following hypertension but does not cause the hypertension. Rarely in pregnancy may be seen a case of pyelonephritis with such marked destruction of the kidney as to bring about high blood pressure and renal failure. Herrick's discussion concludes with the belief that the role of infection in the urinary tract is of no more importance in hypertensive toxemia of pregnancy than it is in cardiovascular disease in general, and that such a role is subsidiary and not fundamental.

Along this line there is an interesting paper by Hayes (20), in which he reports thirty-nine cases of pernicious vomiting, preeclampsia or eclampsia treated prior to delivery by ureteral catheterization, with immediate improvement after kidney drainage, all of which ultimately recovered. Here will be found more evidence pointing to urinary back pressure and stasis causing or at least precipitating toxemias in pregnancy.

Crabtree (5) reports a case of inflammatory stricture of the ureter during pyelitis in pregnancy. After stricture formation, kidney function on that side was lost, the blood pressure became elevated and a diagnosis of toxemia was made. An operation was performed and the kidney and ureter were removed. A study revealed that chronic pyelonephritis and subacute glomerulonephritis were present, probably subsequent to ureteral stricture.

Longcope (29) opens an interesting field of investigation in his observations on chronic pyelonephritis and its association with hypertension. The whole process may start with an infection of the urinary tract during pregnancy. If the woman suffers the usual symptoms over a period of time, the condition is usually considered to be a chronic pyelitis. It is not until headache, nausea, vomiting, loss of weight, lassitude, dyspnea, or, in some instances, convulsions occur that the patient, in a more or less desperate condition, is discovered to have more than a pyelitis. The blood pressure is not often elevated during the early stages of the disease. As the disease advances it is usual to find a rise in the blood pressure; the specific gravity of the urine is low and finally becomes fixed; albumin, rarely casts or red blood cells, but many leukocytes are found in the urine, and in most cases the culture of the urine gives at some time a growth of B. coli. A striking feature is the unexpected frequency with which a marked impairment of renal function occurs at a time when the patient feels perfectly well. Months or years before the terminal stage the nonprotein nitrogen of the blood may be distinctly elevated and the urea clearance far below normal.

One of the most important evidences of the disease is obtained by pyelography. The pyelogram shows dilated and chronically infected, and flattened pelves. There may be uniform or irregular dilatation of the ureters. Arteriolar sclerosis or diffuse glomerular nephritis is superimposed upon the renal structure. Longcope thinks the majority of cases of chronic pyelonephritis originate from hematogenous infections from the gastro-intestinal tract on previously healthy urinary tract tissue, but it is quite possible that some form of obstruction to both ureters may predispose the kidneys to infection as, for instance, the enlarged uterus of pregnancy.

With regard to the hypertension and renal damage he concludes that the hypertension which was found to exist in the terminal stages of the disease, or was observed to develop during the course, was not associated with pronounced arteriosclerosis of the vessels of the kidneys or with widespread uniform involvement of the arterioles of other organs. The height of blood pressure, both systolic and diastolic, changed considerably from time to time in many of these patients. These findings are often present in patients who are suffering from the form of hypertension designated as essential. Further, he quotes the experimental work of Goldblatt and his coworkers who have shown that ischemia of the renal tissues of dogs and monkeys, produced by reducing the arterial flow of blood to the kidney through constriction of the renal arteries, results in persistent elevation of blood pressure. Longcope feels that the analogy of these experiments to that of hypertension and chronic pyelonephritis is very suggestive.

Christian (43) also believes that a considerable number of patients who have had pyelonephritis may progress slowly with an insidious form of nephritis and the terminal picture one sees with glomerular nephritis.

There is, of course, much that is unknown surrounding the nephritides, the toxemias and hypertension, but such observations as have been made on the kidneys and ureters of pregnancy with reference to these diseases bring us a trifle closer to their real nature.

The value of urological methods in the diagnosis and treatment of urinary tract infection, with or without pregnancy, should be fully realized by urologists and obstetricians alike. A urological department would, therefore, seem to be an essential part of every up-to-date maternity hospital (1).

SUMMARY

The author has attempted to show that pyelitis in pregnancy is a frequent complication of that state; that the pregnant woman is extremely susceptible to infection in her urinary tract because of the physiological changes occurring; that the pathology is quite variable in extent and severity, depending on several factors, so that infection frequently persists even years after delivery; that unless recognized early and treated adequately serious permanent damage will result from the infection, including hydronephrosis, chronic pyelitis, tubular

and glomerular damage to the kidney, hypertension, and possibly pregnancy toxemias.

CONCLUSIONS

1. Renal damage resulting from pyelitis in pregnancy is an aspect of this disease which has only recently been investigated, as far as can be learned from the literature.

2. The incidence of pyelitis in pregnancy varies rather widely in published reports, ranging from 1 per cent to 25 per cent.

3. The physiological dilatation of the ureters, together with pressure from the growing uterus cause urinary stasis which is the important predisposing cause of pyelitis.

4. Pyelitis can be mild or severe. The renal damage resulting will depend on the severity of the infection, previous urinary infection, repeated pregnancies, and whether treatment has been adequately and promptly employed.

5. Antenatal pyelitis is cured completely in from 32 per cent to 73 per cent, depending on the authors quoted. Cure during pregnancy is rare.

Postnatal pyelitis is cured completely in from 61 per cent to 100 per cent of the cases. The majority are cured in from twelve to eighteen months after delivery.

6. Bacteriuria persists after pregnancy in most cases and is very likely to cause pyelitis in subsequent pregnancies, unless the infection is cleared with adequate treatment.

7. In the cases which are not cured one finds periureteritis, hydroureter, hydronephrosis, pyonephrosis or even destruction of glomeruli and tubules of the kidney with abscess formation, contracted kidney, glomerular fibrosis and vascular thickening.

8. The most recent work on this subject strongly suggests an etiological relation of pyelitis to the toxemias of pregnancy and hypertension.

9. The importance of prompt and adequate urological diagnosis and treatment in urinary infections, with or without pregnancy, cannot be overemphasized.

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PROCEEDINGS
of the
University of Maryland Biological Society

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ONE-HUNDREDTH PROGRAM MEETING

The Centennial Program Meeting and Dinner of the University of Maryland Biological Society was held on November 14, 1939. Dinner was served at 7:15 P.M. in the dining room of the University Hospital, with the past presidents of the Society as guests of honor. At 8:30 P.M., Mr. O. G. Harne, President of the Society, opened the program meeting in Gordon Wilson Memorial Hall and introduced the past presidents of the Society, each one standing in recognition of his name.

Dr. George L. Streeter, Director of the Department of Embryology, Carnegie Institution of Washington, who was the guest speaker, read a paper on "The Origin of the Idea of the Three Germ Layers". This was followed by discussion and a lantern slide demonstration of the present concepts of embryology.

An abstract of Dr. Streeter's paper appears below.

ORIGIN OF THE IDEA OF THE THREE GERM LAYERS*†

George L. Streeter

A summary given of ideas concerning the initiation of life and the beginning of the embryo as taught by the ancients and the scholars of the Middle Ages. The first statement of the three germ layer theory and its elaboration into its present-day form

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* From the Department of Embryology, Carnegie Institution of Washington, Baltimore, Md.

† Read at the meeting of the University of Maryland Biological Society, November 14, 1939.

was outlined. By means of lantern slides a demonstration was given of the first steps in development as they occur in primates (macaque embryos), showing that the segregation of the materials of the ovum is primarily a matter of cell cleavage and thereby the auxiliary substances of the ovum are set apart from the formative cells proper. This constitutes the initial chapter in the development of the embryo. Then follows the segregative individuality of cell masses and cell tissues with their inductive interactions upon each other. In such terms an improved understanding of development appears to be at hand and in this functional and more realistic manner of analysis the three germ layer concept can be largely disregarded.

ONE-HUNDRED-AND-FIRST PROGRAM MEETING

The annual business meeting of the University of Maryland Biological Society was held in Gordon Wilson Memorial Hall on December 12, 1939 prior to the Program Meeting. In addition to the President of the Society, Mr. O. G. Harne, the following members were present: Doctors Carr, Figge, Beck, Hartung, McCrea, Gittinger, Amberson, Oster, Forman and Dobbs, and Mr. W. Harne.

The reports of the Secretary, Dr. Edward C. Dobbs, and the Treasurer, Dr. C. Jelleff Carr, were read and unanimously accepted.

Dr. Walter H. Hartung, Chairman of the Committee on Nominations, read the following list of nominees:

President: Dr. C. Jelleff Carr
Secretary: Dr. Thomas Grubb
Treasurer: Dr. M. W. McCrea
Councillor for one year: Dr. Frank J. H. Figge
Councillor for four years: Dr. Edward C. Dobbs

These nominees were unanimously elected.

The following candidates were elected to membership in the Society:

Full membership:
- Dr. William Hahn, School of Dentistry
- Dr. John F. Lutz, School of Medicine
- Mr. Fred Ellis, School of Medicine
- Dr. W. L. Hard, College Park, Maryland

For associate membership:
- Miss Mildred Donohue, School of Medicine
- Miss Grace Zimmerman, School of Medicine

The speaker for the Program Meeting was Dr. Harvey G. Beck, Professor of Clinical Medicine, School of Medicine, University of Maryland. The topic of his paper was "Carbon Monoxide as a Domestic Hazard". Dr. Beck presented the results of clinical research on patients from the University Hospital and from the diagnostic clinic, showing the acute and chronic symptoms of carbon monoxide poisoning. This subject was very interesting and greatly enjoyed by the members and guests who were present.

At 10:30 P.M. the meeting was adjourned by the newly elected President, Dr. Carr.

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The names listed above are officers for the term beginning July 1, 1939 and ending June 30, 1940.

A LETTER FROM THE ACTING DEAN CONCERNING MEDICAL ALUMNI ASSOCIATION SCHOLARSHIPS

Dear Fellow Alumnus:

At my first meeting with the Board of Directors of the Medical Alumni Association plans were discussed for the promotion of scholarships for poor boys who come to us for admission. After it had been explained that we lose each year a number of these boys, who are very acceptable from all standpoints, because we lack sufficient scholarship aid for Freshmen, the Treasurer of the Association moved the donation of $1,200.00 to support a scholarship of $300.00 a year for four years, and I was instructed to proceed with the selection of an acacceptable applicant.

An applicant was selected and a searching investigation was made of him by the Committee on Admissions. On December 5 the candidate came to Baltimore, was interviewed by the six members of our Committee on Admissions and by four officers of the Medical Alumni Association. At the Alumni Association meeting that night, the candidate's scholastic credentials were presented. The ten interview reports were all favorable and the candidate was accepted for scholarship support for four years, provided he maintains a satisfactory scholarship record. This boy was accepted for admission and will be with us in September, 1940.

On December 9, I received the $1,200.00 from the Medical Alumni

Association. On the same day I secured permission from the Secretary-Treasurer of the Trustees of the Endowment Fund of the University of Maryland to set up an earmarked savings bank fund to be known as "The University of Maryland Medical Alumni Association Scholarship Fund". The money from the fund, thus protected, may be withdrawn only on a joint order signed by the Treasurer of the Medical Alumni Association and the Chairman of the Committee on Admissions, which must be sent to the Secretary-Treasurer of the Trustees of the Endowment Fund, who, in turn, orders the disbursement. This service is gratis.

At the Alumni Association meeting on December 5 it was felt that the Association had taken a step forward in promoting this type of support and it decided to concentrate its efforts chiefly along this line from now on. It was suggested that there are a great number of our Alumni who would become much more interested in the activities of the Alumni Association now that we have decided to support this worthwhile program.

For each unit of $300.00 it would be possible to help one excellent student through Medical School and thus contribute to society one good physician. Incidentally, for each student of high rank accepted, the Committee on Admissions would eliminate one less desirable student; this would raise the scholarship standing of each entering class proportionately.

Each year the Committee on Admissions has to turn away earnest youngsters of excellent background, personality and achievement, because we lack sufficient scholarship aid. Consider the beneficial effect the admission of ten students of this caliber would have on the scholarship and wholesomeness of each entering class; there will be no trouble in securing these students once we announce the availability of this support in our annual catalog.

At present there are sufficient supporting members of the Medical Alumni Association to take care of its operating expenses. Each one hundred new members will provide for an additional scholarship. However, contributions may be made directly to this Scholarship Fund.

The Medical Alumni Association is to be commended on the adoption of this progressive program and will receive wide support in its promotion.

Judging from the interest expressed and the support proffered by those to whom I have explained this program, it will not be long before we shall have an annual income of about $3,000.00 to use for this purpose.

Those of us who have taken part in setting up this first scholarship have derived so much pleasure from it and look forward with such keen interest in following the yearly advancement of this first boy, that I felt all of our Alumni would like to know something about this new activity of our Medical Alumni Association.

Very sincerely yours,
H. Boyd Wylie, (Signed)
Professor of Biochemistry
and Acting Dean

ITEMS

Dr. Clyde A. Clapp, Professor of Ophthalmology, presented a paper on "Some Ophthalmological Problems in relation to Medicine and Surgery" before the Bridgeport Medical Society of Bridgeport, Connecticut on November 9, 1939.

Dr. Thomas F. Vestal (U. of Md. 1929), resident physician at the Worcester County Sanatorium, Worcester, Massachusetts, has accepted the position of Director of the Division of Industrial Hygiene in the State Department of Health at Raleigh, North Carolina.

Plans for the Sixth Annual Luncheon-Meeting of the New England Alumni, which was to be held at Worcester last June in conjunction with the annual meeting of the Massachusetts Medical Society, did not materialize. However, it is expected that the series of meetings will be resumed in June, 1940 when the Massachusetts Medical Society meets in Boston. This annual gathering of the New England Alumni always has been welcomed, with a goodly proportion of the 500 graduates of the three Baltimore schools in attendance.

Dr. O. Costa Mandry (U. of Md. 1921) was elected President of the Puerto Rico Medical Association for the year 1940.

On November 8, 1939, Dr. L. A. M. Krause, Associate Professor of Medicine, spoke at the meeting of the Cordell Historical Society held in Gordon Wilson Memorial Hall. The subject of his address was "Some Interesting Descriptions of Disease in the Bible."

Doctor Charles E. Gill (U. of Md. 1927), State District Health Officer for the Massachusetts Department of Public Health with headquarters at Westfield, Massachusetts, has resigned to accept the position of Deputy State-County Health Officer in Prince George County, Maryland, with headquarters at Upper Marlboro. Since 1934 Doctor Gill has been secretary-treasurer of the Massachusetts and New England Alumni and has assisted in the five annual luncheon-meetings held in Massachusetts in connection with the annual meetings of the Massachusetts Medical Society.

Dr. Maurice Feldman, Assistant Professor of Gastro-Enterology, was one of the speakers at the first meeting of the Maryland Academy of Medicine and Surgery held on October 12 at the Belvedere Hotel. His topic was "Aneurysms of the Abdominal Aorta."

Rocky Mountain spotted fever, which has claimed increasing numbers of victims in the East in the last several years, was the topic of discussion at the meeting of the Medical and Chirurgical Faculty on November 3, 1939. Dr. T. Nelson Carey, Associate Professor of Medicine, spoke on the clinical aspects of the disease and illustrated his address with colored lantern slides of the subject.

Dr. Irving Freeman (U. of Md. 1935) announces the opening of an office for the practice of internal medicine at 2420 Eutaw Place.

The following surgeons have been certified by the Board of Orthopedic Surgery:

- Dr. Allen F. Voshell
- Dr. Albertus Cotton
- Dr. Moses Gellman
- Dr. John V. Hopkins
- Dr. Harry L. Rogers
- Dr. Henry F. Ullrich

Dr. Moses Gellman, Associate Professor of Orthopedic Surgery, has been appointed Chairman of the Orthopedic Section, Baltimore City Medical Society, for 1939–40. He has also been elected a member of the American Academy of Orthopedic Surgeons.

Dr. John V. Hopkins was appointed Surgical Director of the United States Fidelity and Guaranty Company in July, 1939.

Dr. Henry F. Ullrich, Associate in Surgery and Orthopedic Surgery, was elected to the Interurban Orthopedic Society in 1938 and received the degree of Doctor of Science from the American College of Physicians in 1939. In April, 1939, Dr. Ullrich spoke before the Frederick County Medical Society on "Differential Diagnosis of Joint Lesions." A paper on "Operative Treatment of Scoliosis" by Dr. Ullrich appeared in the August, 1939 issue of the American Journal of Surgery.

Dr. Allen F. Voshell, Professor of Orthopedic Surgery, has recently published the following papers:

"Regeneration of the Lateral Condyle of the Humerus after Excision." Published in the Journal of Bone and Joint Surgery, April, 1939.

"Treatment of Poliomyelitis during Postparalytic Stage." Published in the Medical Record, November 15, 1939. (Read before the Fifteenth Annual Meeting of the Academy of Physical Medicine, Washington, D. C., in 1938.)

"Important Features in the Treatment of Fractures." Published in the Ohio State Medical Journal, September, 1939. (Read before the Ninety-third Annual Meeting of the Ohio State Medical Society, Toledo, Ohio in May, 1939.)

"Fractures of the Shaft of the Femur Treated by Ambulatory Method." Now in process of publication. (Read as part of symposium before the American Orthopedic Association, Buffalo, N. Y. in June, 1939.)

In addition Dr. Voshell has made the following addresses:

"Crippled Children in Maryland and the United States," Rotary Club of Denton, Md., November, 1939.

"Discussion of Fracture Fundamentals and Errors," Medical Society of Ohio County, West Virginia, November, 1939.

"Oft-forgotten Facts about Fractures," Medical Society of Columbia, South Carolina, September, 1939.

OBITUARIES

ROBERT PARKE BAY, M.D.

Dr. Robert Parke Bay was born at Pylesville, Harford County, Maryland, on November 8, 1884, the son of Bertha Parke Bay and Daniel A. Bay. He received his early education at the county public schools and from private tutors, following which he entered the University of Maryland and matriculated at the School of Medicine in 1901. He graduated with honors in 1905.

Dr. Bay was a major in the Medical Corps of the Maryland National Guards, receiving his commission in 1911 from the late Governor Austin L. Crowthers, and served in the World War. Through 1904–1905 he was a resident student at the University Hospital, surgical interne in 1905–1906, and resident in medicine 1906–1907. He was resident surgeon at Bay View Hospital, now Baltimore City Hospitals, in 1907–1908, and returned to the University Hospital as medical superintendent in 1908 and held that position until 1910. He was connected with the teaching staff of the medical school and held the rank of Associate in Surgery, and Associate Professor of Clinical Surgery; in 1933 he was appointed Professor of Oral Surgery. In 1910 Dr. Bay was appointed Instructor of Oral Surgery in the School of Dentistry of the University of Maryland, and in 1916 was made Professor of Oral Surgery. He was elected in 1919 to the Faculty Council of Dentistry. In 1914 the State Industrial Accident Commission was created to administer the Workmen's Compensation Law of Maryland, which law went into effect that year. Governor Phillips Lee Goldsborough, then Governor of the State of Maryland, appointed Dr. Bay as Medical Adviser of that Commission, which position he held throughout the years until his death. He became Chief Surgeon of the Maryland General Hospital in 1932 and was Visiting Surgeon to a number of other hospitals in Baltimore City and those at Havre de Grace, Elkton, and Montgomery County, Maryland. He had held the position of Chairman of the Board of City Charities and was interested in innumerable civic activities. In 1936 he became the President of the Baltimore City Medical Society, having served as Vice-President of that organization the preceding year. He served as a member of the Board of Censors and was also on the Honor Committee of the Medical and Chirurgical Faculty of Maryland. He was a member of the American College of Surgeons, American Board of Surgery, Baltimore City Medical Society, Southern Medical Association, Medical and Chirurgical Faculty of Maryland, Maryland Club and Baltimore Country

Dr. Robert Parke Bay

Club. He wrote a number of papers on medical subjects and talked frequently before county and state societies.

With his scientific training and experience was combined a rare sympathy and common sense that made him outstanding in his profession. Early in his practice he began the training of younger men. This became a definite hobby and a great satisfaction to him. The young men who were fortunate in becoming his assistants can testify to his absolute mastery of surgery, his skillful fingers, and the correctness of his judgment. His interest in these individuals continued uninterruptedly even after they became practitioners, and the loyalty they displayed in his judgment and to him as a man is worthy of recordation. He was never too busy to give his sane, logical advice to young students, nor to homesick and footsore young nurses who looked upon him as their adviser and revered him. His absolute dependability, his cheerful attitude and the tender gentleness that the sick recognized as real endeared him with bonds of affection more lasting than can be expressed. Dr. Bay was a man's man, a good sport, a raconteur of much charm, with a legion of friends in all walks of life. His warm hospitality and outstanding personality always dominated his presence. He stretched out a helping hand to all in need and gave not only of his time and his funds, but of that shining inner ray that made his charities a living index of his character. His interest mainly was his profession, but he too was a man who keenly enjoyed the great outdoors, spending the summers at his cottage at Ocean City, Maryland. He was an excellent shot at clay pigeons, headed the Oriole Gun Club for a number of years and, on one occasion, was the Maryland State doubles champion. He was an enthusiast of both field and wild fowl shooting and a great lover of dogs. In later years he became a golf enthusiast.

In 1916 he married Nancy M. McNabb of Harford County, a graduate of the School of Nursing of the University of Maryland, who survives him.

Dr. Bay died suddenly in the early morning of January 1, 1940, the result of a cerebral hemorrhage evidenced but a few hours before. In his death the medical fraternity of the State has lost a leader, a not unworthy successor of the great names of the past which have made the medical fraternity of Maryland illustrious throughout the nation; and the people of Maryland have lost a great citizen.

CHARLES A. REIFSCHNEIDER.

SYDNEY M. CONE, A.B., M.D.

Dr. Sydney M. Cone, formerly Associate Professor of Pathology of the School of Medicine, University of Maryland, died on December 19, 1939, following a prolonged illness.

Dr. Cone, a son of Herman and Helen Guggenheimer Cone, was born at Jonesboro, Tennessee on November 16, 1869. He obtained the degree of Bachelor of Arts from Johns Hopkins University in

Sydney M. Cone, M.D.

1890 and was graduated from the School of Medicine, University of Pennsylvania, in 1893. Upon his return to Baltimore he served as an intern in the Johns Hopkins Hospital.

While an intern, under the influence of Welch and Halstead, he became interested in pathology. For a time he was active in the practice of orthopedic surgery, but derived his greatest happiness from his research and teaching in pathology.

In 1895, 1896 and 1900, Dr. Cone took post graduate courses at the Universities of Heidelberg, Strasbourg and Vienna. During this period he published an article entitled "A Case of Carcinoma Metastasis in Bone from a Primary Tumor of the Prostate." He was thus among the first to call attention to the now well-recognized tendency of prostatic carcinoma to metastasize to bone.

He was Professor of Pathology at the Baltimore Medical College from 1905 to 1911 and was a valued member of the staff of the Department of Pathology at the University of Maryland from 1916 to 1938.

In May, 1917, Dr. Cone, as a member of the Medical Officers Reserve Corps, was sent to England with an orthopedic unit. He remained there for two years, spending the greater part of this time at the Alder Hey Hospital in Liverpool. Although listed as a surgeon he soon became interested in the histologic study of war injured nerves and devoted much of his time to the laboratory. As a result of his studies he devised a new method of staining nerve tissue, which he designated as the "neurokeratin stain". He also published a number of articles on the pathology of peripheral nerve injuries. In recognition of his meritorious services in the United States Army he was promoted to the rank of major.

Dr. Cone was always basically a pathologist. The influence of his work in orthopedic surgery, however, can be seen in his publications, for the majority of his numerous writings in pathology were the result of his studies of lesions in bone. While a member of the staff of the Department of Pathology he undertook the laborious task of studying sections of ribs from routine autopsies in an attempt to correlate osseous changes with those in other tissues. At the time of his retirement he had in his laboratory portions of ribs from hundreds of autopsies.

As a teacher of histopathology Dr. Cone possessed unusual ability. He was patient yet firm, sympathetic yet meticulously thorough.

Dr. Cone was a kindly, retiring person and those who knew him found in him a sincere friend. His wise counsel, his spirit of tolerance and his friendly cooperation are sorely missed by his associates.

Hugh R. Spencer.

WILLIAM L. HAMMERSLEY, M.D.

Dr. William L. Hammersley, B.M.C. 1898, aged 70, died on July 17, 1939 at the Methodist Hospital in Indianapolis, Indiana of heart disease.

Dr. Hammersley was born on December 5, 1868 at West Lafayette, Ohio, where he lived until he was twenty-three years of age. Thereafter he attended Athens and Mount Union Colleges in Ohio. Later he taught school for a while and then entered the Baltimore Medical College. After receiving his doctor's degree he settled in Frankfort, Indiana where he established a practice.

In addition to a large private practice Dr. Hammersley was physician and surgeon for the Pennsylvania Railroad. He took a personal interest in helping the younger generation by sending a pharmacist, a minister and three nurses, as well as his own two sons, through school. One son now is a physician, the other a dentist.

Dr. Hammersley had been in practice in Frankfort, Indiana for the past forty years, where he was widely known as an outstanding surgeon and humanitarian. His passing will be greatly mourned by his many patients and friends.

MILFORD LEVY, M.D.

Dr. Milford Levy, P. & S. 1915, aged 47, died on October 10, 1939, at his home in Pikesville, Maryland, after an illness of several months.

Dr. Levy was born on January 20, 1892, the son of Aaron and Hannah Levy, at Tallahassee, Florida. He received his primary and secondary education there and in Baltimore, and graduated from the College of Physicians and Surgeons in 1915. Following internships at the Sinai and Mercy Hospitals in Baltimore, and the Montefiore Hospital in New York City, he entered the United States Army and served as a captain in the Medical Corps, Department of Nervous and Mental Diseases, from 1917 to 1919. After the war he continued his study of neurology and psychiatry at the Postgraduate School of Medicine, University of Pennsylvania. From 1921 to 1927 he was neurologist and psychiatrist to the Federal Board of Vocational Education, later the Veterans' Bureau; and since 1927 he was consultant in neurology and psychiatry to the United States Veterans' Bureau. At the same time he practiced his specialty ably and successfully in Baltimore, and served on the staffs of the Mercy, Sinai, St. Agnes, South Baltimore General and University Hospitals. Since 1921 he has been associated with the neuropsychiatric clinic at Mercy Hospital, and with the Department of Neurology of the University of Maryland, where he held the position of Associate Professor in Neurology at the time of his death. He was a member of the American Medical Association, American Psychiatric Association, Southern Medical Association, Society for Research in Nervous and Mental Diseases, the Baltimore City Medical Society, and the Big Brother League of Baltimore.

Dr. Levy had achieved a most enviable position in his chosen field of work. He was a man whose character and ability were manifested by the respect and affection of his many friends, patients, associates and students. His untimely death removes from the Balti-

Milford Levy, M.D.

more medical profession a brilliant and able neurologist and psychiatrist, and from his associates a staunch, genial and loyal friend.

Philip F. Lerner

GEORGE LOUIS VIEWEG, SR., M.D.

Dr. George Louis Vieweg, Sr., P. & S. 1902, died at his home in Wheeling, West Virginia, on November 30, 1939 of valvular heart disease.

Dr. Vieweg was born in South Wheeling, where he received his preparatory education. After graduating from the College of Physicians and Surgeons he practiced medicine in Baltimore for many years. Later he moved to Wheeling, West Virginia, where he was a member of the staff of two local hospitals.

He served as a captain in the United States Medical Corps during the World War and was stationed at a base hospital in Montgomery, Alabama.

Dr. Vieweg was affiliated with the County, State and American Medical Associations and had served as a medical director of the Ohio County Tuberculosis Sanatorium for a number of years. He had been active in Masonry.

Bankes, Charles W., Middleport, Pa.; P. & S., class of 1881; aged 82; died, August 27, 1939, of cerebral hemorrhage.

Bishop, Berton Mell, Archer, Fla.; P. & S., class of 1890; aged 70; died, August 22, 1939, of organic heart disease.

Carroll, J. J., Holyoke, Mass.; class of 1905; aged 61; died, November 19, 1939.

Carter, George William, Alexandria, Va.; class of 1878; aged 84; died, July 20, 1939, in Jacksonville, Fla., of uremia and hypertrophy of the prostate.

Carter, Merville H., Baltimore, Md.; P. & S., class of 1878; aged 81; died, September 5, 1939, of cerebral hemorrhage, arteriosclerosis and hypertension.

Cohn, Michael A., San Diego, Calif.; P. & S., class of 1893; aged 71, died, July 10, 1939, at La Jolla, Calif.

Earhart, John H. T., Westminster, Md.; class of 1888; aged 77; died, September 10, 1939, of coronary thrombosis.

Fleich, Oscar, Brooklyn, N. Y.; B.M.C., class of 1897; died, June 4, 1939.

Friedlaender, Bernhard, Detroit, Mich.; B.M.C., class of 1898; aged 68; died, August 14, 1939, in Rochester, Minnesota, of thrombosis of the mesenteric vein, gangrene of the intestine and peritonitis.

Fritz, Gustave Adolph, Baltimore, Md.; class of 1915; aged 48; died, July 9, 1939, of heart disease.

Harrington, Michael W., Springfield, Mass.; B.M.C., class of 1901; aged 66; a member of the American Medical Association and Massachusetts Medical Society, and a member and recent president of the Springfield Academy of Medicine; died, November 12, 1939.

Hertzman, Carl O., Ashland, Wis.; P. & S., class of 1900; aged 72; died, July 25, 1939, of chronic nephritis and uremia.

Jones, Hiram C., Logan, W. Va.; P. & S., class of 1889; aged 78; died, September 28, 1939, in the Huntington Orthopedic Hospital.

Long, Henry Fletcher, Statesville, N. C.; class of 1892; surgeon to a hospital bearing his name; aged 70; died, July 28, 1939, of hypostatic pneumonia and erysipelas.

Lowry, Willis Edward, Sr., Laredo, Texas; P. & S., class of 1892; aged 69; died, July 29, 1939, in the Santa Rosa Infirmary, San Antonio.

Mendelsohn, Lewis, Jersey City, N. J.; B.M.C., class of 1901; aged 66; died, August 26, 1939, of pulmonary embolism.

Morrill, Jenness, Falkland, N. C.; class of 1888; aged 74; died, September 9, 1939, of chronic endocarditis, mitral insufficiency and chronic interstitial nephritis.

Neal, George A., South West Harbor, Maine; aged 67; died, September 17, 1939.

Noland, Benjamin Franklin, Bassett, Va.; P. & S., class of 1881; aged 86; died, October 20, 1939, of cardiac failure.

Parlett, Edgar Merryman, San Francisco, Calif.; B.M.C., class of 1902; aged 59; died, August 21, 1939.

Rankin, Barrick Samuel, Kingwood, W. Va.; B.M.C., class of 1907; served during the World War; aged 67; was drowned, August 22, 1939.

Rees, Rees Bynon, Bakersfield, Calif.; class of 1900; owner of a hospital bearing his name; aged 71; died, August 19, 1939, of coronary embolus, adenoma of the prostate and diabetes mellitus.

Salinger, David, Chicago, Ill.; class of 1894; aged 80; died, September 17, 1939, of cerebral hemorrhage and arteriosclerosis.

Salles, John M., New Bedford, Mass.; B.M.C., class of 1911; aged 56; died, November 26, 1939.

Sexton, C. Hector, Dunn, N. C.; class of 1890; aged 83; died, August 30, 1939, of coronary sclerosis.

Shemwell, Joseph F., Baltimore, Md.; class of 1889; aged 75; died, June 25, 1939.

Sorell, Alfred Merritt, Baltimore, Md.; P. & S., class of 1907; aged 57; died, July 21, 1939, of pyelonephrosis.

Tull, Harry C., Salisbury, Md.; class of 1900; aged 65; died, July 17, 1939, in Ocean City, of coronary thrombosis.

West, Marshall, B., Catonsville, Md.; class of 1901; aged 62; died, June 9, 1939, of hypertensive cardiovascular disease.

Williams, Thornton Riggs, Bala-Cynwyd, Pa.; P. & S., class of 1882; aged 88; died, June 23, 1939.

Whiteside, Robert B., Lott, Texas; P. & S., class of 1889; aged 73; died, July 28, 1939, of cerebral hemorrhage and arteriosclerosis.

Zellars, William A., Shaker Heights, Ohio; P. & S., class of 1891; aged 73; died, August 11, 1939, in Cleveland, of uremia.

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FACTORS CONCERNED WITH RENAL STONE FORMATION*†

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INTRODUCTION

Along with the improvements in methods of diagnosis of conditions in the urinary tract have come the development of drugs and other therapeutic measures possessing a specific effect on certain organisms that infect this tract. These facts coupled with many new researches relative to the etiology of renal calculi have brought about the necessity for a more careful scrutiny of the part bacteria play in stone formation.

The factors most commonly associated with the formation of renal calculi are as follows: (1) bacteria, (2) urostasis, (3) colloid-crystalloid imbalance, (4) metabolistic disturbances, (5) vitamin A deficiencies, (6) hyperparathyroid influences, (7) bone injuries and bone diseases, and (8) Randall's "calcium plaque" theory.

Some of these conditions may occur singly or they may occur together, some may be primarily the cause of the stone or merely contributory. Infections seem to play an important part in some instances, while in others infections cannot be found. John Hellstrom (3) says, "Undoubtedly there are numerous different factors which more or less favor the appearance of calculi and these factors need not be the same in every case of lithiasis." To quote Higgins (1), "From experimental and chemical observation, it appears evident that no one etiological agent is entirely responsible for the formation of all kidney stones." Randall (2) thinks that over 50 per cent of the cases of renal stones fail to present clinical evidences as to why they occur.

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* From the department of Genito-Urinary Surgery, School of Medicine, University of Maryland.

† Read before the Philadelphia Urological Society, December 18, 1939.

It is well to keep in mind all during this discussion that, at least in some instances, these factors are manifestations of calculus disease and that the stone is not just a foreign body in the kidney.

In order to discuss bacteria concerned with renal stones it is necessary also to discuss the many factors collaborating in their formation. It is a very confusing subject. Many men who have spent their lives working on problems of bacteria and stone differ among themselves in interpreting their results. However, certain facts stand out regardless of the real etiology and it is known that bacteria associated with renal calculi are of important significance.

It has yet to be proved that bacteria are the cause of renal calculi. At the same time, that there is a definite relationship between renal infections as demonstrated in urine smears and cultures, and some of the types of renal calculi, there is no doubt.

For practical purposes renal stones are divided into primary and secondary stones, depending on whether or not they are infected. This division is unsatisfactory because many primary stones are found to be accompanied by infection or are shortly secondarily infected, if not at once.

Primary stones are chiefly those that are precipitated in acid and neutral urine and consist of uric acid, urate and oxalate. The secondary calculi are composed of magnesium ammonium phosphate, potassium carbonate and ammonium urate; these are generally found associated with heavy infection and in alkaline urine.

MECHANICS OF STONE FORMATION

According to Swift Joly (4), three conditions must be present before a stone can form. First, there must be a deposit of crystals in the urinary passages, or, at least, the crystalloid must be in a continued state of supersaturation. Second, minute calculi must be retained within the urinary tract. Third, the colloids of the urine must be coagulated into the form of a *gel* and not remain in a finely divided suspension or *sol*.

When the colloid is in the form of a gel, because of some unknown change in the urine, its surface tension becomes reduced and a layer of insoluble crystalloid covers its surface. The layer of fluid next to the colloid becomes grossly supersaturated, so that crystals tend to form in and around the gel.

Such a mass of colloid and crystals grows by the apposition of fresh colloid which, in turn, attracts fresh crystals. The mass continues to increase in size and forms the nucleus of a developing calculus. The irritation of this stone nucleus produces a serous exudate. The colloid of this exudate becomes precipitated on the nucleus and soon covers any asperities on its surface, only to attract more crystals as the stone continues its growth. Thus the laminated structure can be explained by a continuous and uniform deposit of crystals, coupled with a rhythmically waxing and waning amount of colloid. The origin of stone is, then, caused by a failure of the colloid mechanism, but its nature and chemical composition depend largely on the character of the diet. Therefore, in every stone formation colloids and crystalloids are indispensable. In support of this it is pointed out that no one has ever succeeded in producing a calculus in a test tube. Lichtwitz (5) found that normal urine contains about 0.83 grams of colloid per liter. The amount depends on diet and is increased when the protein content is high. It is also increased in acute diseases, such as pneumonia, and in acute or chronic inflammations of the kidney.

In 1916 Neuberg defined certain substances in the urine which were almost insoluble in water, that could easily be dissolved in aqueous solutions of other salts, particularly in solutions of sodium benzoate, sodium hippurate, sodium salicylate and sodium mandelate. He called this property hydrotropy.

Snapper (6) has shown that salicylates, hippurates and mandelates prevent the precipitation in vitro of colloidal solutions of fibrinogen and sodium nucleate with aluminum sulphate. Since the presence of hippuric acid has been demonstrated in normal human urine, these authors think that this substance may counteract the formation of calculi in two ways, (1) by stabilizing the urinary colloids, and (2) by increasing the solubility of calcium salts. This experimental work has possibilities of extreme importance in the therapeusis of renal calculi.

INFECTION

The ways in which a bacterial infection can favor stone formation are as follows: (1) increase the pH of the urine so that a precipitation of phosphates occurs; (2) split the urea molecule with the formation of insoluble magnesium ammonium phosphate; (3) form a foreign body on which crystals may be deposited; (4) alter the state of the colloid from a suspension to a gel, and (5) give rise to a tissue exudate which contains a considerable amount of foreign colloids.

It is possible that several of these factors acting together are necessary for the genesis of a calculus (4).

Rydgaard of Copenhagen (8) found that primary urinary calculi generally consist of calcium oxalate and, as a rule, are sterile, and that secondary calculi occur in alkaline media and are produced experimentally by A-avitaminosis, and further that they are always attended by infection. Rydgaard concludes that various investigations and facts seem to indicate that something else, not yet ascertainable, is necessary for the formation of stone in addition to supersaturation of the urine, which is an indispensable factor in the formation of every calculus. He advances the proposal that this unknown cause may be damage to the kidney itself.

Many years ago Rovsing found that 21 per cent of all recurrent stones in the kidney were infected and that the organism associated with the stone had the power of splitting urea. This observation of Rovsing's had been neglected until recently, when it became apparent that this power to split urea is all-important.

Formerly the Micrococcus ureae was considered to be the only organism that split urea; now it is known that many organisms possess this power. From a knowledge of chemistry one observes, however, that the fermentation of urea gives rise to ammonium carbonate. This is split up, the ammonia combining with the magnesium and phosphoric acid to form triple phosphates, while the carbonate combines with the calcium to form calcium carbonate.

Ever since the time Rosenow and Meisser (7) were able to produce stones experimentally with streptococci obtained from the teeth of stone-bearing patients, more efforts have been made to reproduce stones experimentally. Keyser (15) has been successful in duplicating Rosenow's experiments with the streptococcus. Hager and Magath (9) were more successful than Hryntschak of Vienna (10) in producing stones with the proteus, but recently he has succeeded in producing calculi in rabbits by injecting cultures of staphylococci intravenously. His previous method was to produce a certain degree of obstruction in one ureter by tying a ligature loosely around it. In 80 per cent of his experiments the animals developed calculi in the obstructed kidney. Hryntschak also points out that staphylococci have the power of coagulating or precipitating albuminous colloids and that it is possible that this power has something to do with the formation of calculi.

Apparently, however, no one has succeeded in reproducing stone in the urinary tract with the colon bacillus, and yet this organism occasionally has the power of splitting urea.

According to general experience, it is especially infections with urea-splitting bacteria, particularly staphylococcus and B. proteus, which lead to lithiasis in the urinary passages. B. coli is thought to be subordinate or secondary.

For fifteen years John Hellstrom of Stockholm (3) has been observing the relationship between staphylococci and the so-called primary or sterile stones. He has come to the conclusion after these years of observation that staphylococci are the most important cause of the occurrence of calculi in connection with infections of the urinary tract. Time and time again in the presence of persistently sterile urine Hellstrom was able to find the matrix of these supposedly sterile calculi composed of pure staphylococci. He has dissolved the inorganic substance of calculi by means of hydrochloric acid, and then taken smears of the resulting gelatinous matrix. In old, long-standing cases the organisms from the central portions have lost the power of retaining the Gram stain, but those nearer the periphery were deeply stained by this method. In other cases he embedded the organic stroma in paraffin and made serial sections. The cocci were seen "lying in concentric layers and between them an amorphous thready substance." In yet other cases he succeeded in culturing cocci from the central portions of the calculi. Hellstrom, therefore, believes that staphylococci are in many instances the cause of renal calculi.

Infections of the kidney with the staphylococcus are commonly seen following suppurative lesions of the skin, such as furuncles, carbuncles, etc., as well as infections in other parts of the body. These infections usually involve the cortex and may progress to form a carbuncle of the kidney, or may subside and in so doing infect the pelvis. Sir Girling Ball (11) was one of the first to draw attention to this pathologic process and intimates the possibility of the cocci being an etiological factor in stone formation.

Hellstrom found that these "staphylococcic stones," as he terms them, are composed of calcium carbonate, triple phosphate, amorphous phosphates, and in a few instances a small amount of calcium oxalate. These staphylococcic stones were found in alkaline urine and the staphylococci were found to have the power of splitting urea.

If Hellstrom is enthusiastic about cocci being the etiological agent in stone formation and since he found an alkaline urine in all of his cases, one wonders what his ideas are of the etiology of stones found in acid urine. Further, in his series of 750 cases of renal and ureteral calculi at the Maria Hospital in Stockholm, only 22 per cent were infected, but of these 22 per cent he found 75 per cent were staphylococcic infections. Discussing Hellstrom's statistics, Randall (2) cultured seventy-five impacted ureteral stones at open operation, instead of urine cultures cystoscopically. Randall found 52 per cent were infected. Lett of London (12) found staphylococci in more than one-half of 740 cases of urinary calculi.

In the Mayo series, 20 per cent of the infections were caused by staphylococci and practically all took a positive Gram stain. In Higgins' (1) recent series of 200 recurrent calculi, coexisting renal infection was present in 81.5 per cent, with the staphylococcus as the predominating organism. In the series of Brown and Earlam of Australia (13), 40 per cent were staphylococci, all of which have the power of splitting urea. Stadnichenko studied thirty strains of gram-positive cocci isolated from his cases of pyelitis and found fourteen cases which split urea.

The Proteus bacillus is the most powerful urea-splitter of all the bacteria and is found associated with stone next in frequency to the cocci. The stones with this infection are large, they disintegrate easily, and generally contain an abundance of ammonium urate. Magnesium ammonium phosphate and calcium carbonate are present in large quantities in the urine of patients infected with urea-splitting organisms. These stones are notorious for their frequency of recurrence, because the proteus is most resistant to therapy. Braasch (20) says that of all the organisms in their experience at the Mayo Clinic, the B. proteus is the most difficult to eradicate. This organism produces a deep seated purulent infection in the kidney with much pathological change in the pelvis and ureters.

The other organisms commonly found in renal calculi are the Aerobacter aerogenes, Pseudomonas, Salmonella, Shigella and Alcaligenes; except for the Proteus, they all usually respond to therapy in a similar manner. They are found with stones and are frequently characterized by a urea-splitting property. Their presence may be suspected by the repeated finding of a high pH in the urine.

In Grossmann's (16), Lett's (12), Joly's (4) and Hryntschak's (10) large series, the colon bacillus was found most frequently in the infections that occurred with renal lithiasis. They attached little importance to the colon bacillus as a factor in stone formation in spite of its frequency. In fact, Rovsing claims that some strains of B. coli have a destructive action on stones already formed. Kretchmer says that while the common cause of renal infections *is* the colon bacillus, the largest number of infections *are not associated with stone.* This type of infection, when found alone and uncomplicated, usually limits its pathological attack to the superficial structure of the urinary tract.

Some authors, for example, Cyranka and G. de Illyes, believe that large coral stones are found in infections with B. coli and think that some strains of B. coli appear to contribute directly to the appearance of stones. Certainly according to Schneider and Keyser (15), B. coli can alter the shape of oxalate crystals in the urine. Streptococci faecalis was found by Braasch (20) to be almost always a secondary invader in infections of the kidney and rarely found unless a nephrostomy tube was in place.

Whether or not micrococci are pathogenic is open to question; they are classified as saprophytic.

It has been pretty well proved that organisms do not pass through the kidney without causing a pathological condition.

The presence of anaerobic bacteria in stone cases is rare.

The organisms found in the urine of stone cases are usually considered to be secondary infections; however, these organisms may have taken no part in the *genesis* of the calculus. It has long been known that while bacteria may be found in calculi, they must be identified in the *nucleus* before one can assume that they have any influence in creating stones.

It seems generally accepted, however, that stones occurring with infection grow more quickly than aseptic stones.

Foci of infections in the teeth, tonsils, cervix uteri or prostate will frequently be a factor in producing renal infections, but as a cause of stone formation little can be found in the literature in spite of Rosenow's experiment. Higgins found no apparent relationship between organisms cultured from foci of infection and those cultured from the urine.

Grossmann (16) considers that infections in the upper urinary tract often arise from a postgonorrheal infection of the prostate gland and seminal vesicles. He is of the opinion that out of 350 cases of infection with stone, thirty-seven were of this type, the offending organism being usually the staphylococcus. Joly (4) reports that he has seen similar cases.

CHEMICAL COMPOSITION

In Higgins' series of recurrent stone he found that when the composition of stones was caused by uric acid, urates or oxalates, the urine was usually sterile; whereas, with infection, the composition was almost always phosphates, carbonates, and only traces of oxalates. In other words, uric acid, urates and oxalates are the most common constituents of calculi originating in uninfected urine, while the stones caused by a hypervitaminosis are composed of calcium phosphates. This salt is the most common constituent of all calculi originating in infected urine, but distinctly does not occur apart from infection (4).

Greta Hammarsten (17) has found that calcium oxalate calculi (the hardest stones) are formed "without the cooperation of bacteria." These stones may be formed experimentally in rats in acid, neutral and basic urines. Care was taken during her experiments to keep the urine sterile. She was able to produce calculi in 309 out of 900 rats by diets. Out of these 309 rats that formed stones in this way, only twelve were infected and the infections occurred in stones consisting of magnesium ammonium phosphate, which, of course, produced alkaline urine.

Many of the Continental European urologists have noticed that since the World War there has been a decided "stone wave," presumably because of dietary restrictions occasioned by this war. In children, where the incidence of stone was disappearing before the war there was noticed an increase,—all on an A-avitaminosis basis.

Also during the war Paul (18) found organisms in the urine of eighteen out of twenty cases of lithiasis following compound fractures. This factor of renal calculosis with or without infection has been observed not infrequently in bone immobilizations and prolonged periods of bed rest enforced by other diseases, notably tuberculosis.

In stones, calcium phosphates formed as a result of hyperparathyroid disease, the incidence of infection is not imposing; the fact is noted, however, that parathyroid hyperfunction cannot give rise to oxalate or uric acid lithiasis.

Congenital anomalies of the urinary tract predispose to infection and stone formation. There are not any statistics available to tell of the relationship between the two, but undoubtedly urostasis precipitates both the infection and the calculus.

In his recent work in estimating calcium and phosphorus excretion in the urine, Flocks (19) stated that in thirty-five consecutive cases of stones in the kidneys and ureters all had slight infections of the urinary tract. However, none of them had outspoken urinary sepsis. The organisms cultured were Staphylococcus aureus and Staphylococcus albus, B. proteus, B. coli, and Staphylococcus hemolyticus.

Flocks noted that when there was slight kidney damage there was an increase in calcium concentration in the urine in contrast to a diminished concentration in cases of marked destruction of the kidney.

Randall (2) has opened a fresh chapter in the etiology of lithiasis by his observations on the "calcium plaques" that are occasionally seen on renal papillae. These deposits of lime salts are described by Randall as existing in the subepithelial tissues of the papilla and not in the collecting tubules. Randall does not consider these calcium deposits to be inflammatory in origin, since in only 3.9 per cent of his reported cases did he find evidence of tissue destruction, or round cell infiltration. Randall has further demonstrated on these tiny papillary plaques composed of calcium carbonate and calcium phosphate, the genesis of stones formed of a different salt, calcium oxalate. And so, for the first time, one of the earliest forms of stone formation is described by this observer. He, therefore, does not believe that the etiology of stone is totally dependent on local infection. After his experiences on animals with bacterial toxins, he says: "An acute renal insult of such toxic nature, or the kidneys' reaction to a low grade but oft repeated toxic insult, can be directly associated with tubular and intertubular calcium deposition, similar in every way to that which we have observed in man."

PERSONAL EXPERIENCES

During the last five year period at the University Hospital in Baltimore there have been 110 cases of renal calculi, or an incidence of 0.3 per cent of the general admissions of 34,712. This compares with the London series of Lett (13), whose incidence was about 1 per cent in nearly 275,000 admissions. Stone incidence in some parts of this country runs as high as 3 to 4 per cent of the general hospital admissions. Therefore, the incidence at the University Hospital is small.

In this series of cases the stone occurrences in the ages from one to twenty are few. This reduction of stone incidence in children agrees with observations elsewhere. It is considered to be the result of the wider range of diet that children now have and the better management of their renal infections. Most of the cases in this series occurred in the twenty to forty year period, as is usual. For some reason or other there was quite a high incidence, twenty-six cases, between the ages of sixty and seventy.

It was possible to culture but fifty-eight of 110 cases, out of which eighteen were negative. The B. coli was found in eighteen cases, in four of which it was mixed with other organisms. The other organisms were Staphylococcus albus—fourteen, B. proteus—three, Alcaligenes—three, Aerobacter and Shigella—one each.

This compares with Gilbert Smith's recent statistics as follows: B. coli either alone or mixed in seventeen out of thirty cases, five cases were sterile, B. proteus in thirteen, Staphylococcus in six, and pyocyaneus in one.

Chute reported from the Massachusetts General Hospital that the following organisms split urea: B. proteus, Staphylococci, Non-hemolytic Streptococcus, B. pyocyaneus, B. influenzae, and Micrococcus.

In his series the author found the above organisms to split urea and to be associated with stones, with the exception of the Micrococcus flavus and influenza which were not encountered. He had also, four Aerobacters, three Alcaligenes and one Shigella, which were found with renal stones and which split urea.

The technique employed at the University Hospital has been to make a stained smear of the urinary sediment in every case of renal stone. It is of importance to examine the freshly voided specimen in a sterile container. This is particularly true of infections with urea-splitting organisms, when the character of the urine may be considerably changed after standing for only an hour or so at room temperature. Not infrequently the bladder urine was found to be alkaline and the kidney specimens acid. It is of importance to determine the pH of the urine from each kidney.

Simple staining of the urinary sediment by the Gram method will tell whether or not organisms are present and will divide them into bacilli and cocci; this will also produce the gram-positive and gram-negative classifications.

The pH of the urine frequently gives a clue to the type of organism and character of the stone. Maslow's statistics are as follows: oxalates average pH 5.9, urates pH 5.6, phosphates pH 6.2, uric acid pH 6.5. Under varying influences, principally the alteration of the pH of the urine, many organisms change their morphological and cultural characteristics so that it is possible to identify an organism on one examination that on another occasion may seem to be, and probably is, an entirely different bacterium.

Cultures from each colony grown must be made.

SUMMARY

1. This discussion is made with the idea of presenting some of the confusion that exists in the relationship of bacteria to renal stones.
2. It is evident that while bacteria may be the etiology of stone formation in some instances, certainly other factors are of equal import.
3. To quote Higgins, "From experimental and chemical observation, it appears evident that no one etiological agent is entirely responsible for the formation of all kidney stones."
4. Randall thinks that over 50 per cent of the cases of renal stones fail to present clinical evidences as to why they occur.

5. The two most common urea-splitting organisms are the Staphylococcus and B. proteus. Therefore, these organisms are regularly found with stones and in alkaline urine. These are the secondary stones.

6. The primary stones are usually found in acid urine, but more and more are organisms found with these stones. In fact, many urologists believe that infection occurs in practically all "sterile stones."

7. The colloid-crystalloid balance in the urine is regularly affected in infections and its disturbance initiates stone formation.

8. In certain instances bacteria change their cultural and morphological characteristics, making them difficult to identify.

9. Organisms that split urea are intimately identified with stone formation, and to prevent recurrences of stones these infections must be sterilized.

10. The B. proteus is the most difficult organism to eradicate from the urinary tract.

11. The staphylococci which formerly were considered a relatively innocuous finding in urine, undoubtedly split urea. They have the power of coagulating or precipitating albuminous colloids and are evidently a considerable factor in stone formation.

12. Calcium phosphate is the most common constituent of all calculi originating in infected urine, but does not occur in stones found in sterile urine.

13. Emphasis should be placed on *slight* renal damage rather than gross destruction in the relationship between infection and calculi formation.

14. Diet affects materially the chemistry of all stones.

15. Apparently relationship of prostatic infection and stone in the kidney is not sufficiently known.

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THE VITAMINS IN OBSTETRICS*†

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In this presentation it is far from the author's intention to become technical or to enter into any detailed discussion of the vitamins, but rather to review briefly some of the known facts about their use in obstetrics and to cite some personal impressions. A great deal of experimental work with the vitamins is still in progress and many of the statements about them must of necessity be guarded.

Vitamin A does not appear to play a particularly important rôle, although it has been stated that a deficiency might interfere with ovulation and that an adequate supply in the body is an excellent prophylactic against puerperal infection. It is also felt to be essential to the intra-uterine growth of the fetus and its continued development for the first year of its extra-uterine life. There appears no reason why this vitamin in maintenance dosage of 6,000 to 12,000 units daily should not be given during pregnancy if indicated, but on the other hand proof seems insufficient to warrant its routine administration.

Vitamin B_1 and the B complex are quite important, and many minor and some major complications have been ascribed to their deficiency. It is doubted that this is entirely true, yet the fact that such a deficiency may make certain conditions worse must constantly be kept in mind. B_1 appears quite necessary for the proper metabolism of the carbohydrates, a lack of it causing the process to cease with the formation of pyruvic acid, a product which, while it does not appear to be toxic, has no nutritive value. Therefore, in hyperemesis, where logically there would be a lack of all of the vitamins, the administration of B_1 in 8,000 units b.i.d. together with the glucose which has come to be accepted generally as a part of the treatment, would seem theoretically justified. Actually, in the author's experience it appears to be of immense value, hastening recovery and stimulating the appetite, and it has been included as a definite part of the treatment. This has been in effect for about one year and, while it may be a coincidence, the fact remains that it has not been necessary to do a therapeutic abortion for hyperemesis in approximately 4,000 admissions to two different clinics. The administration may be by vein in the more severe cases or by mouth in the milder ones, both routes giving satisfaction. Two cases were treated with thiamin chloride alone, in the hope that it might be more properly evaluated as a therapeutic agent. In both instances the vomiting stopped promptly and the patient went on to an uneventful recovery; however, it was felt that the psychic element had not been sufficiently eliminated to warrant drawing definite conclusions. At the present time it is the feeling of everyone in the Department of Obstetrics that B_1 should be used in hyperemesis.

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Results in the late toxemias have not been at all favorable and little evidence has been found to support the contention that a lack of this particular vitamin might be considered as an etiological factor here. As a therapeutic agent it has proved of practically no value, which is quite disappointing.

In those patients showing evidence of nerve involvement as a lack of vitamin B, its administration has proved most satisfactory. Many of the patients with the milder forms of neuritis respond quickly, and the occasional case in the more advanced stage is likewise greatly benefited. One such patient was seen about a year ago, who, among other things, had an almost complete atelectasis of the left lung as the result of unilateral muscular weakness. This cleared up rather quickly under the B complex, she went to term without incident, and was delivered of a healthy, normal baby.

It has been claimed that more than one-half of all pregnant women show a more or less decided lack of vitamin C. When one thinks of the importance of an adequate supply of vitamin C to the individual, and reads of experiments tending to prove that about one-half of C-deficient animals do not conceive, and of those which do become pregnant one-half abort, it is difficult to correlate these statements with clinical experience. It is likely that such claims are true in certain cases. However, it is the author's impression that the pregnant woman usually obtains a sufficient amount of vitamin C in her regular diet to meet her daily needs, and the routine administration of an additional amount is not justified. Where evidences of deficiency are present, it should undoubtedly be given, the dosage being about 400 units daily. The dosage varies with the apparent need of the patient, prophylactically being one capsule three times daily, and therapeutically four or five times this amount.

It appears futile to speak at any length of vitamin D, its value being well recognized and its routine administration with calcium and phosphorus during pregnancy being widespread. It might be well to remember that a large calcium-phosphorus intake with vitamin D does not of necessity insure against dental caries, and patients should still be instructed to visit their dentists regularly.

Much work has been done on vitamin E in the past few years, work with which all obstetricians are quite familiar. It will suffice to say at this time that the author's experience is in accord with that of others. He feels that several pregnancies have been carried to term with the help of this vitamin, which probably would have otherwise aborted. The author has not had any great success in the treatment of abruptio placentae, but this may possibly be because of insufficient dosage.

This brings one to a consideration of the latest of the vitamins to be used clinically in obstetrics, vitamin K, and since most of the work on it is quite recent it will be discussed somewhat more freely than most of the others. Indeed, most of the work is so recent that it should be considered to be still in the experimental stage and definite statements about it avoided.

The principal use of vitamin K, which has in the past been obtained largely from alfalfa but which at this time is or, at least, very shortly will be available as a synthetic product, would appear to be to increase the prothrombin content of the blood when this is lowered, and thus to reduce the tendency toward bleeding. There is no proof that vitamin K is a component part of the prothrombin molecule. It may be that it is essential for the proper function of the tissues manufacturing prothrombin, the principal source apparently being the liver, for when this organ is badly damaged vitamin K therapy has no effect. It seems to have no value when the prothrombin content is normal or nearly so, and its principal use in the adult is in cases of obstructive jaundice accompanied by a hemorrhagic tendency, when it is given in conjunction with bile salts. However, repeated estimations on the blood of the newborn seem to prove rather conclusively that the prothrombin content is almost routinely low, frequently well below the safe limit. Further, it would seem that in premature babies it is usually lower than in term infants and more often approaches the danger point. This may, in part at least, explain the greater frequency and severity of intracranial and other hemorrhages in these infants.

Having determined that the prothrombin content was well below normal in the newborn, the next step was to determine whether it could be raised by the administration of vitamin K, and in one quite recent experiment it was given to a fairly large series of babies whose prothrombin content had been previously determined. It was found that in every instance the content was very definitely and gratifyingly raised, while in a control group of babies who did not receive vitamin K it remained low for the period of observation. Therefore, it might be concluded that vitamin K was responsible for the increase. Two more questions, however, remained to be answered: first, does vitamin K do any damage to the newborn premature or full term infant, and, second, does the prothrombin increase result in a decrease in the bleeding and clotting time? A careful and honest evaluation of the experiments and close observation of the babies would seem to indicate that the first question can be answered in the negative, but the work is too recent and the number of cases too small for anyone to be dogmatic. Certainly there seems no reason why the work should not be continued. In answer to the second question, again one cannot be positive, but there are many reasons to believe that the bleeding and clotting time are quickly and materially reduced following K administration, and everyone who has used this measure at all has been struck by the sometimes marvelous results obtained. A three day old baby bleeding from the nose, mouth and rectum was given 1 cc. (1,000 units) of vitamin K by mouth in the evening and ½ cc. the following morning. By noon all bleeding had ceased; it did not recur, and recovery was uneventful. The bleeding and clotting time of another baby was found to be very much prolonged, up to thirty and twenty to twenty-five minutes. It received one dose of vitamin K and the following day the time for both was well within normal limits, three to four and four minutes.

The next forward step in vitamin K therapy would seem to be in its administration. When given to the mother prior to delivery it is transmitted rapidly to the fetus and appears to cause a higher prothrombin content than when given directly to the baby. The latest time when desired results can be obtained and the dosage necessary have not been determined definitely, but it would seem that if the mother is given a minimum of 2,000 units at least four hours prior to delivery, the baby will be sufficiently protected. The amount may be larger and the time longer, the above being the safe limit in the light of present knowledge.

Is there any benefit to be derived from the routine prenatal administration of vitamin K? No one knows the answer to this question today; however, several series are being studied at this time, and after one or two years one should have a fairly good idea. In the University Hospital Clinic the practice of routinely giving vitamin K to every woman entering the hospital in premature labor was recently introduced. It was planned to compare the fetal mortality, when a sufficient number of cases have been collected, with a like number in which the agent was not used.

One does not wish to appear too enthusiastic about this extremely recent addition to obstetrical therapy and admit that time may prove all or a large part of the above to be false. In the meantime, however, it looks as if vitamin K offers the best treatment available at present in hemorrhagic disease of the newborn, and in the prophylaxis and treatment of multiple, minute hemorrhages in the brain. The bleeding and clotting time of the newborn should be determined before attempting any operative procedure. If found to be prolonged, vitamin K should be given for at least twenty-four hours and the tests repeated before the operation is undertaken.

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ECLAMPSIA*†

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The conservative treatment of eclampsia is well recognized and followed in the majority of the larger clinics today; however, there is no established and universally accepted routine as each clinic has found it necessary to adapt the treatment to its particular organization and facilities. Indeed, it is doubtful that any fixed routine would ever be successful, for in the light of our present knowledge of eclampsia the treatment is an ever-changing one, and procedures which yesterday seemed rational and desirable may tomorrow prove fallacious and unnecessary. A suggestion from this source, an improvement of a more or less established routine from that one, the discarding of old and useless measures, and the addition of new and seemingly desirable ones, should all go to make up the treatment until such time as the cause is discovered. With this purpose in mind the cases for the past five years at the University Hospital are here presented from the standpoint of treatment and results.

Since Stroganoff's original paper appeared there has been a gradual transition from immediate delivery by major operative procedures, either by the abdominal or vaginal route, to the watchful waiting and hands-off policy which is a part of the therapy of the present day. Not until 1934 did this Clinic adopt an absolute conservative treatment with a definite routine. Prior to this date the majority of cases were conducted by the Stroganoff method or one of its many modifications, and a small minority were subjected to radical treatment Through this stage the present routine was evolved and adopted, which is as follows: as soon as the diagnosis of eclampsia is made, irrespective of the type, the patient is given ¼ gr. morphine hypodermically, blood is withdrawn for chemical analysis, and 250 cc. of 25 per cent glucose together with 4 cc. of 50 per cent magnesium sulphate is given intravenously. Following this, 5 or 6 drams of paraldehyde in aromatic elixir is given orally if the patient is awake and able to swallow, or 8 to 10 drams in olive oil is administered by rectum if she is still comatose. The patient is either admitted to or now transferred to a quiet room and a senior medical student is kept in constant attendance, his duty being to keep an hourly blood pressure chart and protect the patient from injury should any further convulsions occur. Frequent visits are made by the nursing supervisor of the delivery suite, members of the house staff, and the visiting obstetrician. This careful supervision is continued without interruption until all danger of further convulsions has passed.

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† From the Department of Obstetrics, School of Medicine, University of Maryland.

As soon as the sedation has reached the point of maximum effectiveness, which requires about one hour, further studies of the case are begun. These include eye ground examination and analysis of a catheterized specimen of urine. Two hours after initial medication the intravenous glucose and magnesium sulphate are repeated; following this only the glucose is given at four hour intervals, and it is continued until such time as the toxemia shows definite improvement. The magnesium sulphate may be repeated again in the first twenty-four hours; however, from Stander's study of the drug no more than 6 gm. can be given safely in one day. Concentrated glucose, 25 per cent, is used in large quantities, between 250 and 300 gm. per day, because a hypoglycemia has been an almost constant blood chemistry finding in all cases of severe preeclampsia and eclampsia at the University Hospital, and this finding is in agreement with Titus who first instituted and recommended this form of therapy. So rapidly utilized is this glucose that even after large amounts have been given the blood sugar remains relatively low, and there appears to be only a slight spill in the urine. Other apparent actions of this concentrated solution which render it valuable are: diuretic, sparing of the liver or possibly actual regeneration of the liver, relief of acidosis, and decrease of cerebral and general edema. No insulin is used as there is no apparent difficulty in metabolizing the sugar.

The fetus benefits by the presence of an available and readily usable food supply. A part of the deplorably high fetal mortality has been ascribed to the existing hypoglycemia.

The morphine is not repeated in the antepartum and intrapartum patients, as a rule, because of its believed detrimental effect on the fetus, but instead the paraldehyde is repeated as frequently as necessary. There is apparently no other drug which has so marked a sedative action without toxic effect to mother or baby as has this one. Cushing, in his pharmacology, states that "Very large quantities of paraldehyde have been taken without fatal results, and in fact without more serious consequences than prolonged unconsciousness." A fall in blood pressure which lasts for several hours is observed in most cases following its administration. A large part of this effect is the result of the hypnotic properties of the drug and possibly a

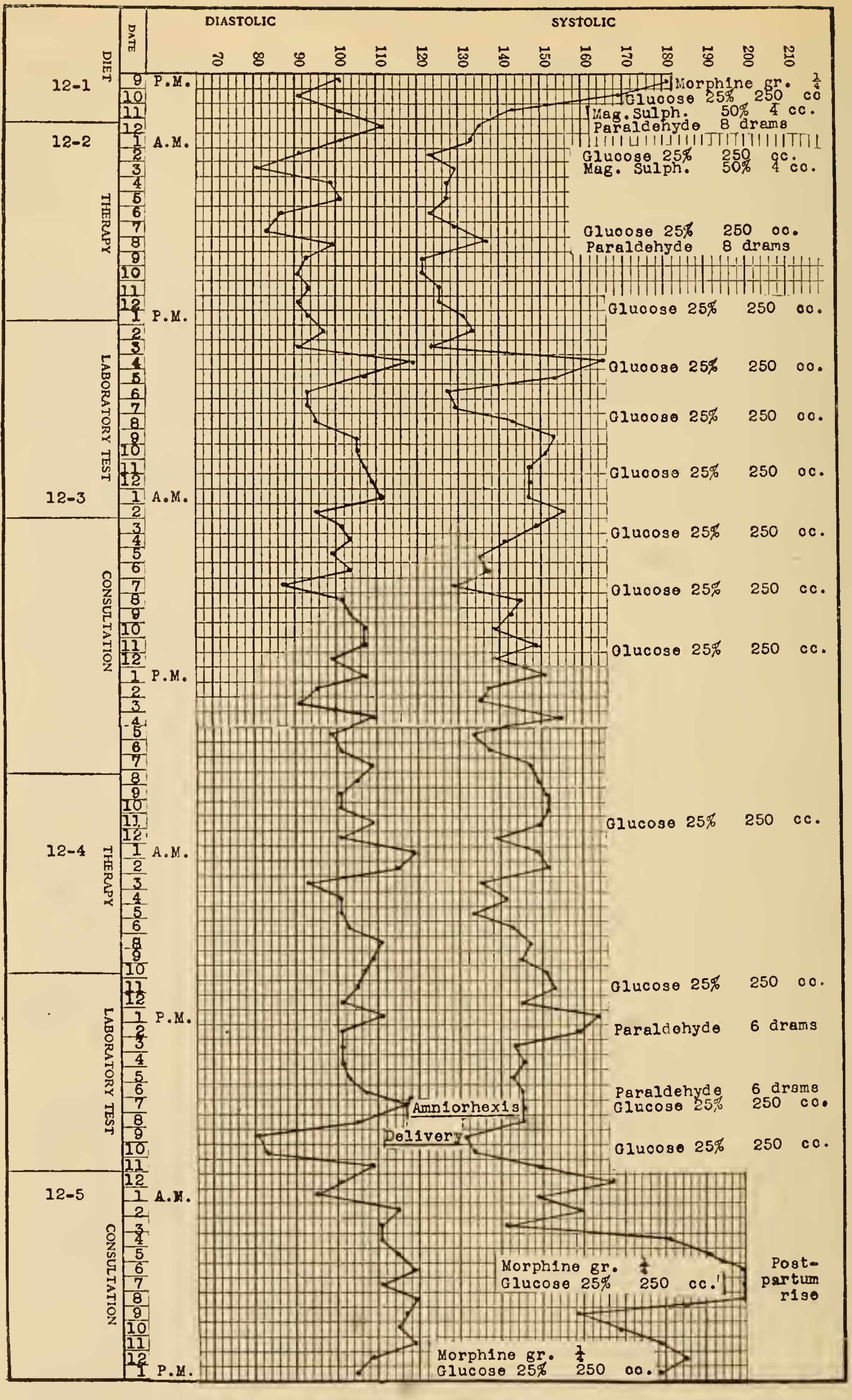

Fig. 1. A blood pressure graph from one of the cases in the series showing the reduction of blood pressure and control of convulsions by the use of paraldehyde, glucose and magnesium sulphate.

small part of its action on the circulatory centers. It does not affect the heart directly, even in very large doses. Concentrated glucose and magnesium sulphate lower the blood pressure temporarily, but when paraldehyde is used in conjunction with them a more desirable result is obtained. The prolonged reduction of arterial tension lasts until the patient begins to react, which is usually after three or four hours. This is demonstrated by a graph from one of the cases in this series.

After a period of twenty-four to forty-eight hours under this treatment of glucose, magnesium sulphate and paraldehyde there are almost always definite signs of improvement, such as less fever, slower pulse rate, reduction of systolic and diastolic pressure, disappearing edema, decreasing albuminuria and returning consciousness. Furthermore, the patient has been free from convulsions for this period of time. There is not a single case in this series which has failed to respond to the extent that convulsions were not controlled.

Up to this point in the treatment there has not been any mention of delivery, and in the conduct of the case the method of delivery is not considered until improvement of the toxemia is definite, or delivery is inevitable. The patient who is admitted in labor or falls into labor after admission receives the treatment described above, and no attempt is made to deliver her until it can be accomplished safely and without difficulty. A primigravida or multigravida who is near or at term and in whom the cervix is fairly well obliterated, with the head dipping or fixed in the pelvis, may be induced by amniorhexis under paraldehyde when the toxemia is subsiding. Having been induced, the patient in labor is watched closely for prodromal symptoms of recurrent convulsions; should they appear, the intensive treatment is begun once more.

The patient with eclampsia whose convulsions occur before the thirty-sixth week of pregnancy is treated for the first forty-eight hours in the routine manner. If, after cessation of convulsions and the return to consciousness the other symptoms do not subside satisfactorily, the condition is considered to be a *posteclampsia* and termination of pregnancy to be indicated. Most of these patients are delivered by the abdominal route. There were two in this report who were treated in this manner, the duration of their pregnancies being thirty and thirty-four weeks respectively. There is, however, an occasional case of eclampsia in early pregnancy that can be carried to term safely.

The anesthetic at the time of delivery is local infiltration, pudendal block or caudal anesthesia. Rarely is an inhalation anesthesia resorted to, and then only nitrous oxide and oxygen are used. General anesthesia is felt to be distinctly contraindicated because a convulsive toxemia is accompanied by a lowered resistance to infection and is usually associated with a mild to a severe degree of pulmonary edema and a marked tendency toward aspiration pneumonia.

To prevent postpartum shock 250 to 500 cc. of 25 per cent glucose is given at the time of delivery and morphine is administered immediately thereafter.

Delivery from below is most frequently accomplished by spontaneous expulsion, low forceps or breech extraction. Episiotomy is used if indicated. Occasionally a midforceps with or without hysterostomatomy is necessary. Abdominal delivery, when indicated, is effected by laparotrachelotomy if possible.

Treatment during the puerperium of antepartum or intrapartum eclampsia consists of a salt-free diet, morphine, and glucose by mouth and intravenously as long as is necessary. Taking the blood pressure frequently and careful watching are also continued until all danger of convulsions has passed. A case of the postpartum type is subjected to the same treatment as those of the ante- and intrapartum types.

Thirty-nine patients, who constitute all of the cases of eclampsia admitted, have been treated in this manner at the University Hospital in the past five years. Of this number seventeen were antepartum, three were ante- and intrapartum, seven were intrapartum, ten were postpartum, and two were ante- and postpartum. The average number of convulsions per patient was 4.7, and the greatest number of any one patient was twenty-six. The majority occurred prior to or immediately after admission.

The incidence as to parity was as follows:

Primigravidae	36
Para 0–0–1–0	2
Para 1–0–0–1	1
	39

Methods of delivery employed were as follows:

Spontaneous	14
Low forceps	19
Midforceps	2
Breech extraction	1
Cesarean section	5
	41 (2 sets of twins)

Operations not including delivery were as follows:

Episiotomy	23
Hysterostomatomy	2
Trachelorrhaphy	1
Proctoperineorrhaphy	2
	28

Two of the cesarean sections performed were for fulminating preeclampsia; despite this treatment one convulsion occurred postpartum in each of these patients. The other three abdominal deliveries were cases that were operated on for posteclampsia because the improvement was only temporary, although convulsions ceased and did not recur. The indications for the two midforceps were occipitoposterior presentation and transverse arrest.

Examination of the complications shows the frequency of infection of any type in these patients, especially those of the chest and generative tract.

Pulmonary		4, or 10%
Bronchopneumonia	2	
Lobar pneumonia and empyema	1	
Partial atelectasis	1	
Generative tract		9, or 23%
Intrapartum	1	
Postpartum	8	

The only other outstanding complications were multiple pregnancy which occurred in two instances, and premature labor which occurred ten times. Postpartum shock and postpartum hemorrhage were witnessed once each.

There were two maternal deaths or a maternal mortality of 5.1 per cent. The first was a 28 year old, Para 0–0–1–0; she had had a normal prenatal course, but when seen at home at the onset of labor her blood pressure was 180/110 and she had a 3 plus albuminuria. She was immediately hospitalized and at this time was given morphine, glucose and magnesium sulphate. The cervix was dilated 1 cm., and the head was in high pelvis. As labor progressed she was given nembutal and paraldehyde analgesia. When the cervix was fully dilated she had an eclamptic convulsion, followed very shortly by a second. After this she remained in deep coma and was delivered by low forceps and episiotomy without any anesthesia. Following delivery she suffered a very mild shock from which she recovered, but her deep coma remained; on the third day postpartum she developed a diffuse bronchopneumonia and died twenty-four hours later without recovering consciousness. No autopsy could be obtained.

The second patient was a 20 year old primigravida admitted a few hours postpartum, having been delivered at home by her own physician. Before admission she had had four convulsions and when admitted was in a profound coma. Her blood pressure was 198/130, urine showed 4 plus albuminuria and many casts, and blood analysis revealed a moderate nitrogen retention. She was quite cyanotic, the pulse was rapid, and respirations were shallow. While treatment was being instituted she had three convulsions in rapid succession, but none thereafter. She developed a massive edema of the lungs and died twenty-four hours after admission. Partial autopsy showed cloudy swelling of the liver and kidneys, an acute splenic tumor and pulmonary edema. The cause of death was postpartum eclampsia and pulmonary edema.

To comment about the first case, it may be said that if a labor of thirty hours could have been forseen for such a fulminating toxemia, a better method of delivery would probably have been cesarean section under local anesthesia. Despite the fact that the patient received ample treatment for the average patient with preeclampsia in labor, she had two convulsions and these were immediately controlled. Instead of recovering from the coma she remained in this state, developed a respiratory infection and died. The second, despite allaying of convulsions by the routine treatment, developed pulmonary edema which in addition to her toxemia caused her death.

The fetal mortality for this series was ten, or 24.4 per cent. The distribution, if the eclampsia is classified according to the time of occurrence of the first convulsion, is as follows:

TYPE	NO. OF CASES	NO. OF BABIES	F.T.L.	F.T.D.	P.L.	P.D.	DEATHS IN LIVE BORN BABIES
Antepartum	22	22	14	2	5	1	2—prematurity—duration of pregnancy—30 and 34 weeks
Intrapartum	7	7	5	2	0	0	1—intracranial hemorrhage
Postpartum twins—2 sets	10	12	6	0	5	1	1—prematurity—32 weeks
	39	41	25	4	10	2	4

There were forty-one babies delivered, which included two sets of twins. There were four full term dead infants. The duration of the pregnancy in the premature dead was twenty-eight and thirty-seven weeks respectively. Three of the four deaths in live born infants were the result of prematurity, the length of the gestation period being thirty, thirty-two and thirty-four weeks; the other death was caused by intracranial hemorrhage.

CONCLUSIONS

1. The series of cases is too small to offer any conclusive evidence of the merits of this form of conservative treatment of eclampsia; however, the results at this time are favorable.
2. Paraldehyde has proved to be an effective and safe drug for controlling eclamptic convulsions.
3. Paraldehyde in conjunction with magnesium sulphate and glucose reduces arterial tension for a relatively long period of time.
4. General anesthesia is contraindicated in eclampsia.
5. Respiratory complications and generative tract infections are the most common and serious sequelae of eclampsia.
6. The maternal mortality and fetal mortality in this series are 5.1 per cent and 24.4 per cent respectively.

STAB WOUNDS OF THE HEART*†

HARRY C. BOWIE, B.S., M.D.

BALTIMORE, MD.

This paper is a discussion of the diagnosis, treatment and complications of stab wounds of the heart and a clinical survey of the cases which have occurred at the University Hospital in the ten year period of 1929–1939.

HISTORY

For years it was thought that operations on the heart were impossible and that should a person be stabbed in this organ the outcome was certain to be fatal. This belief was so widespread among surgeons that even the great Billroth scoffed at the possibility of cardiorrhaphy. Probably the first important observation which made this type of surgery possible was by the Frenchman, Ambroise Paré, who noted that a duelist stabbed in the heart was able to chase his opponent many yards before death occurred. Later Rosenthal and Black were able to prove by animal experimentation that cardiac wounds were not of necessity immediately fatal, that cardiorrhaphy could be performed, and that this procedure improved their chances of recovery. In their work they showed also that there are certain danger zones, namely, the bundle of His, the interventricular septum, and the site of origin of the coronary arteries. Injuries at these points seem to be beyond repair. The first man to suggest cardiorrhaphy was John B. Roberts in 1881. In 1887 Dr. Harvey Reid of Cincinnati sutured a wound in the pericardium, while in 1891 Dalton of St. Louis repeated the procedure with recovery of the patient. In 1896 Farina of Rome performed the first cardiorrhaphy on the human heart. The patient, though, died from pneumonia on the sixth postoperative day. The first successful cardiorrhaphy was done by Rehn of Frankfort in 1896. During the past ten years more cases have been appearing in the literature. This is perhaps the result of an improvement in diagnosis and not an increase in the number of stab wounds.

DIAGNOSIS

When stab wounds of the heart occur, death from hemorrhage may soon follow; however, usually this is not the case. The outcome depends upon two factors, namely, the size of the hole in the heart

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* From the Department of Surgery, School of Medicine, University of Maryland.

† Received for publication February 28, 1940.

and the size of the hole in the pericardium. Most patients have a symptom complex which has been named heart tamponade. It is brought about by bleeding into the pericardial sac and the free blood in turn compressing the heart. The signs and symptoms vary in intensity with the rapidity of the rise in intrapericardial pressure. One author has said that it takes approximately 150 cc. of blood to fill the pericardial sac and produce heart tamponade. The free blood compresses the auricles, mainly because they are thin-walled. Blood is prevented from entering the heart and if it cannot enter it also cannot leave. The situation thus may become more and more acute and, if not relieved, will prove fatal. Death occurs not from hemorrhage but from a stoppage of the heart. The picture is indeed a dramatic one, but not always typical. The outstanding features which usually occur are surgical shock of mild to a severe degree, paradoxical pulse, fall in arterial pressure with a rise in venous pressure, distention of the neck veins, cyanosis, rather severe dyspnea, syncope, a sense of tightness over the chest, fear of suffocation, apprehension and distant heart sounds, and, in extreme cases, anoxemia of the brain with unconsciousness may occur. Cases in which the diagnosis is doubtful should be carefully watched. Precardial aspiration may be done to confirm or disprove the diagnosis. However, in most instances it is unnecessary, except to relieve the tamponade temporarily until pericardotomy can be performed. In these cases, in which the symptom complex has come on slowly and there is doubt as to diagnosis, fluoroscopy is of value. This was brought out by Bigger, who showed that in heart tamponade cardiac pulsations are obliterated and the precardial shadow appears practically immobile. The roentgenograph has proved of little value.

OPERATIVE TREATMENT

Treatment may be carried out either by surgical operation or by repeated aspirations of the precardial cavity. The former is more often the procedure of choice. The operation can be performed under local or general anesthesia and in late cases without any anesthetic at all. The author believes a local anesthetic should be used because in practically all of these cases there is some injury to the lung, convalescence is usually stormy, and the chances of lung complications are increased, especially if a general anesthetic is employed. When local infiltration is used, sufficient preoperative sedation such as morphine should be given.

The method of approach depends on the location and level of the point of entrance of the knife. Probably the quickest and safest incision is the intercostal one, with disarticulation of either one or two cartilages at their junction with the sternum. A portion of the sternum may even be resected if further exposure is deemed necessary. In most cases the pleura has been injured and a hemapneumothorax has resulted, or the pleura may be opened in exposing the pericardium. The sucking which follows may be overcome by packing off the rent with gauze. The internal mammary artery is always a source of worry and hence should be looked for and clamped as soon as it is cut. At times it is so close to the sternal edge that ligature of the ends can be accomplished only by transfixing the sutures. The pericardium in heart tamponade appears as a big, bulging, tight mass lying behind the sternum. The evidence of pulsation is usually slight if present at all. It is of a dark purplish hue, yet glistening. The laceration of the pericardium can be seen readily. This can be picked up by two traction sutures and enlarged so that the heart can be exposed. If the bleeding is coming from the ventricle, it is controlled after the method of Cutler and Beck. In the event the auricle is injured, the bleeding is best controlled by first clamping and suturing around it. Once the tamponade has been relieved and bleeding controlled it is safe to give the patient a transfusion or intravenous fluids. The pericardium may be left open or closed loosely and should not be drained. Should it be drained infection is almost sure to follow. If the pleura is opened it should be closed snugly after the blood has been removed.

COMPLICATIONS

The postoperative course is usually a stormy one. The complications when they occur are divided into early and late. In the early group belong recurrence of tamponade either because the bleeding was not completely controlled, or because a pericardial effusion has formed, or because the pericardium was closed tightly. If the patient is going rapidly downhill and the cause seems to be tamponade, a pericardial tap may be done. Many cases develop subcutaneous emphysema. This is likely to come on soon after operation and usually subsides readily. Other cases, especially the fatal ones, develop a persistent paralytic ileus. Later complications are pleural effusion on the left side and, less frequently, empyema. The former is taken care of by thoracentesis and the latter by pleural drainage. Bronchopneumonia also develops, but occurs just as it may complicate any other operative procedure. Pyopericardium may follow such injuries but is relatively infrequent if the pericardium is not drained. When it does occur the streptococcus hemolyticus is the most common offender. Because of this the use of sulfanilamide postoperatively as a prophylactic measure seems indicated.

CASE SUMMARIES

During the ten year period considered in this paper there have been six cases of stab wounds of the heart diagnosed and operated on at the University Hospital. A short summary of each is as follows:

Case No. 1: B. H., a 25 year old black male, was admitted on November 23, 1929, after having been stabbed with a stiletto in the left chest fifth interspace in the region of the midclavicular line. Pericardotomy was done under general anesthesia. Laceration of the left ventricle was found and sutured, and the pericardium drained. The patient died on November 25, 1929. Autopsy showed a fibrinous pericarditis; the culture was not reported. Operator: Dr. C. Karns.

Case No. 2: G. H., a 34 year old black male, was stabbed with a knife in the left chest in the second interspace, 1½ inches from the midline. He was admitted to the hospital on March 4, 1931, about one-half to three-quarters of an hour after having been stabbed. Immediate pericardotomy was performed. The first portion of the procedure was carried out without any anesthetic whatsoever because he was profoundly unconscious and pulseless. The approach was through a long intercostal incision with disarticulation of the second and third costal cartilages, the ribs then being spread apart. The point of injury of the heart was into the left auricular appendage. This was clamped and then sutured. The pericardium was left open and an airtight closure of the chest wound was made. Débridement of the stab wound was then performed. Once the tamponade was relieved, consciousness returned and he had to be given an anesthetic. Postoperatively, the patient developed an empyema of the left side. This was drained on March 25, 1931. Convalescence was stormy but he was discharged as improved on May 8, 1931. This patient was followed for a number of years; when last seen he was enjoying good health and had completely recovered. Operator: Dr. A. M. Shipley.

Case No. 3: R. K., a 36 year old black male, was admitted on June 27, 1937, one-half hour after having been stabbed with an ice pick in the left chest second interspace at the edge of the sternum. A pericardial tap was performed and blood obtained. Immediate operation followed under general anesthesia. The approach was through a curved incision at the left edge of the sternum with the concavity to the left. The third, fourth and fifth rib ends were resected. No definite bleeding point was found. The blood was aspirated. The pericardium was drained with a rubber tube and the tube was removed the next day. The patient had an uneventful recovery and was discharged as improved on July 18, 1937. Operator: Dr. C. Johnston.

Case No. 4: J. G., a 38 year old black male, was admitted on September 10, 1938, about ten minutes after having been stabbed in the left chest with a pen knife. The point of entrance was in the fourth interspace, 2½ cm. to the left of the midsternal line. Immediate pericardotomy was performed under local anesthesia. The approach was through a long intercostal incision, with disarticulation of the fourth and fifth cartilages at the sternum. The pericardium was aspirated and no definite bleeding point found. A Penrose drain was inserted into the pericardial sac and the wound closed. The postoperative course was a stormy one and the patient died on September 12, 1938. A roentgenograph taken before death showed evidence of bronchopneumonia on the right side. Autopsy revealed a streptococcus pericarditis and a small stab wound of the apex of the heart. Operator: Dr. J. C. Hamrick.

Case No. 5: C. G., a 25 year old black male, was admitted on April 16, 1939, one-half hour after having been stabbed in the left chest with a knife. The point of entrance was the fifth interspace in the midclavicular line. Immediate pericardotomy was performed under local anesthesia. The approach was through a long intercostal incision, with disarticulation of the fourth and fifth costal cartilages from the sternum. A gutter wound of the left ventricle was found which required two sutures. The pericardium was closed loosely and the chest closed without drainage. The patient had an uneventful recovery and was discharged as improved on May 10, 1939. Operator: Dr. J. C. Hamrick.

Case No. 6: P. L., a 24 year old black male, was admitted on September 17, 1939, one and one-half hours after having been stabbed in the left chest with a knife. The point of entrance was the fifth interspace, one inch to the left of the midclavicular line. On admission the patient appeared shocked but had no other typical signs. He improved considerably after his shock was treated but when turned on the right side for exploration of his stab wound in the left flank, he immediately went back into shock and developed a definite paradoxical pulse. Pericardotomy was then performed under local anesthesia. The approach was through a long intercostal incision, with disarticulation of the fifth costal cartilage. A rib spreader was inserted and a gutter wound of the left ventricle found. An apical traction suture was inserted into the tip of the heart and the laceration repaired with five sutures. The pericardium was closed loosely and the chest closed without drainage. The post-operative course was uneventful and he was discharged as improved on October 30, 1939. When last seen on February 28, 1940 he was in good health and was employed as a barber. Operator: Dr. H. C. Bowie.

ANALYSIS OF CASES

In analyzing these cases it is of interest to note that they were all in black males. Four of the stab wounds were from knives, one from a stiletto, and another from an ice pick. Perhaps because of the anxiety which prevailed an accurate description of the clinical picture was not recorded in the first case. The only notations made were shock, syncope and distant heart sounds. The remaining cases showed shock and dyspnea, followed later by syncope and distant heart sounds. The third, fourth and fifth cases showed paradoxical pulses on admission, with distention of the neck veins. The sixth case, however, did not have a definite paradoxical pulse until about one hour after admission and at no time did he have markedly distended neck veins. In this case, the development of the paradoxical pulse when the patient was turned on the right side may be explained as follows: the laceration of the pericardium was on the left lateral surface, and when prone the patient bled into his left pleural cavity; when turned on his right side the laceration was uppermost, the pericardium became filled with blood and tamponade was produced. This occurred so rapidly that there was not time to finish cleansing the stab wound in the left flank for exploration. If this is the mechanism which actually occurred, then it may be a test of value and of diagnostic aid in bringing out the paradoxical pulse, that is, the patient should be turned on the side opposite to the stab wound.

General anesthesia was used in three cases; one died and two recovered. Local anesthesia was also used in three cases, with one death and two recoveries. The local anesthesia was effective, but each patient had one-half grain of morphine sulphate preoperatively.

Four of the stab wounds caused injuries to the left ventricle, one of which went into the heart cavity proper. No bleeding point could be found in one case, and the left auricular appendage was entered in still another.

All cases either had an injury to the left pleura or the pleura was opened because two thoracenteses had to be performed on all except the first and fourth cases, and both of these died.

Empyema developed in the second case, but recovery occurred after pleural drainage.

Because Case No. 4 died of streptococcus pericarditis, the fifth and sixth cases were given sulfanilamide. Whether this aided in their recovery cannot be ascertained.

Of the six cases, two died. The coroners' reports were as follows: Case No. 1: fibrinous pericarditis; congestion of the lungs. Case No. 4: streptococcus pericarditis; small gutter wound of the tip of the left ventricle.

The last three cases were each transfused twice postoperatively, but autotransfusion was not attempted.

The usual treatment of stab wounds of the heart is pericardotomy with cardiorrhaphy; however, Strieder and Singleton have reported two cases that were successfully treated by aspiration. Perhaps Case No. 3 in this series could have been handled satisfactorily by this method.

The mortality in most cases is approximately 50 per cent. In the author's cases it was 40 per cent, but the series was a small one.

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CHRONIC, UNDERMINING, BURROWING ULCERS*†

THURSTON R. ADAMS, M.D.

BALTIMORE, MD.

In 1935 Meleney (1) reported a group of cases of chronic, undermining, burrowing ulcerations which follow wounds, either traumatic or operative. It is caused by a micro-aerophilic hemolytic streptococcus, which as the name implies is an attenuated form of streptococcus that can live and propagate with little or no air present. The onset is gradual and is not accompanied by gangrene of the skin. There is an undermining and burrowing under the skin, mostly confined to the subcutaneous tissues, but at times deep sinuses are formed which extend through muscles and may erode large blood vessels, thus causing death from hemorrhage. The base of the ulceration has a dirty gray gelatinous appearance. Occasionally these areas are quite painful. As the undetermining increases, multiple small areas of ulceration appear.

This infection may occur anywhere on the body. The most common sites have been the abdomen, neck, chest and perineum.

These patients have moderate fever, leukocytosis, and mild secondary anemia, and their morale is usually shattered by its chronicity.

Most all of the antiseptic drugs have been used and none have proved satisfactory. Wide excision of the ulceration has met with equally poor results. Ultraviolet light did not halt the progress of the disease.

At times these infections appear to improve. There is a decrease in the discharge and the pale granulations take on a more healthy appearance, but as a rule this is short-lived and the infection continues to burrow and undermine the tissues.

Meleney (2) in searching for some drug to help these people, tried zinc peroxide. This was decided upon because zinc peroxide liberates oxygen slowly and because he was able to consistently grow an anaerobic hemolytic streptococcus. This drug is not irritating to the tissues.

Zinc peroxide is a fine white powder containing about 50 per cent of the peroxide. It has been prepared commercially by the DuPont Chemical Company of Niagara Falls, N. Y. under the name of Z.P.O.

These ulcerations should be excised as soon as the diagnosis is

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* From the Department of Surgery, School of Medicine, University of Maryland, and the Surgical Service of the University Hospital.

† Received for publication March 1, 1940.

made and the wound packed with a paste made of zinc peroxide. The zinc peroxide should be heated for four hours at 140° C. before using. The paste is made by mixing equal parts of the zinc peroxide and sterile distilled water. This paste should be packed in close contact with the tissues and the whole wound covered with vaseline gauze to keep the zinc peroxide paste from drying. This plan of treatment should be carried out daily until there is definite improvement of the wound and disappearance of the micro-aerophilic hemolytic streptococcus. Sulfanilamide (3) has been used in these infections along with excision and zinc peroxide. It has proved efficacious and should be used provided the patient does not show alarming toxic symptoms from the drug. However, sulfanilamide alone is not sufficient to control the infection.

Following the above therapy, healthy granulation tissue usually appears in a few days. The wound is then treated as any clean, healthy wound, i.e., by skin graft or closure, depending on the area involved.

CASE REPORT

M. F., (No. 23080), a 24 year old, markedly obese white female, was admitted to the University Hospital on March 13, 1939 because of recurrent attacks of right lower quadrant pain, the last attack being nine days prior to admission. An appendicectomy was performed on March 14, 1939 under gas, oxygen and ether without any difficulty. McBurney's incision was used and closed with catgut without drainage. On the second postoperative day the patient's temperature rose to 103°F. Examination of the wound was negative. The temperature came down on the third day and continued to run from 100° to 101°F. On the fifth day the wound was opened and considerable purulent material found. The infection had spread laterally and down to the pubis. Stab wounds were made for dependent drainage and rubber tubes inserted. Cultures showed hemolytic streptococci. Administration of sulfanilamide was begun and a blood concentration of 6 to 9 mg.% was maintained for ten days. During this therapy the temperature ranged from 99° to 100°F. The amount of drainage gradually decreased and the patient was discharged on April 16, 1939. The wounds continued to drain so the patient was rehospitalized (No. 24732) on May 26, 1939. Examination at this time showed an extensive undermining of the abdominal wall extending from the midline around into the flank. Cultures at this time showed an anaerobic hemolytic streptococcus. The wound was now opened widely and curetted thoroughly. Zinc peroxide paste was applied to the wound, being careful to see that it was in contact with all of the tissues. These dressings were changed every three days under anesthesia because the patient's morale was poor and she complained bitterly at any attempt to change her dressings. A slight elevation of temperature continued for about ten days and gradually came to normal. The wound improved and rapidly filled in with healthy granulation tissue. Cultures were no longer positive for the anaerobic hemolytic streptococci. The zinc peroxide therapy was then discontinued and the wound was dakinized for a few days. The skin edges were brought together with interrupted silver wire sutures. The wound was completely healed in September, 1939. There has been no further trouble.

SUMMARY

This case is reported in the hope that the complications and treatment used here may prove of value to others. The writer claims no originality in its recognition or treatment, and refers the reader, for more detailed information, to the extensive treatises of Meleney on the subject. It is offered as a suggestion that in complicated wound infections showing little or no tendency to heal, careful cultures be made for the anaerobic hemolytic streptococcus so that Meleney's specific zinc peroxide therapy can be used.

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POLLEN COUNTS*†‡

HOWARD M. BUBERT, M.D. AND SELMA ROSENBERG, A.B.

BALTIMORE, MD.

For the past two years the authors have presented annually in the *Bulletin* a chart showing results of pollen counts for Baltimore (1, 2).

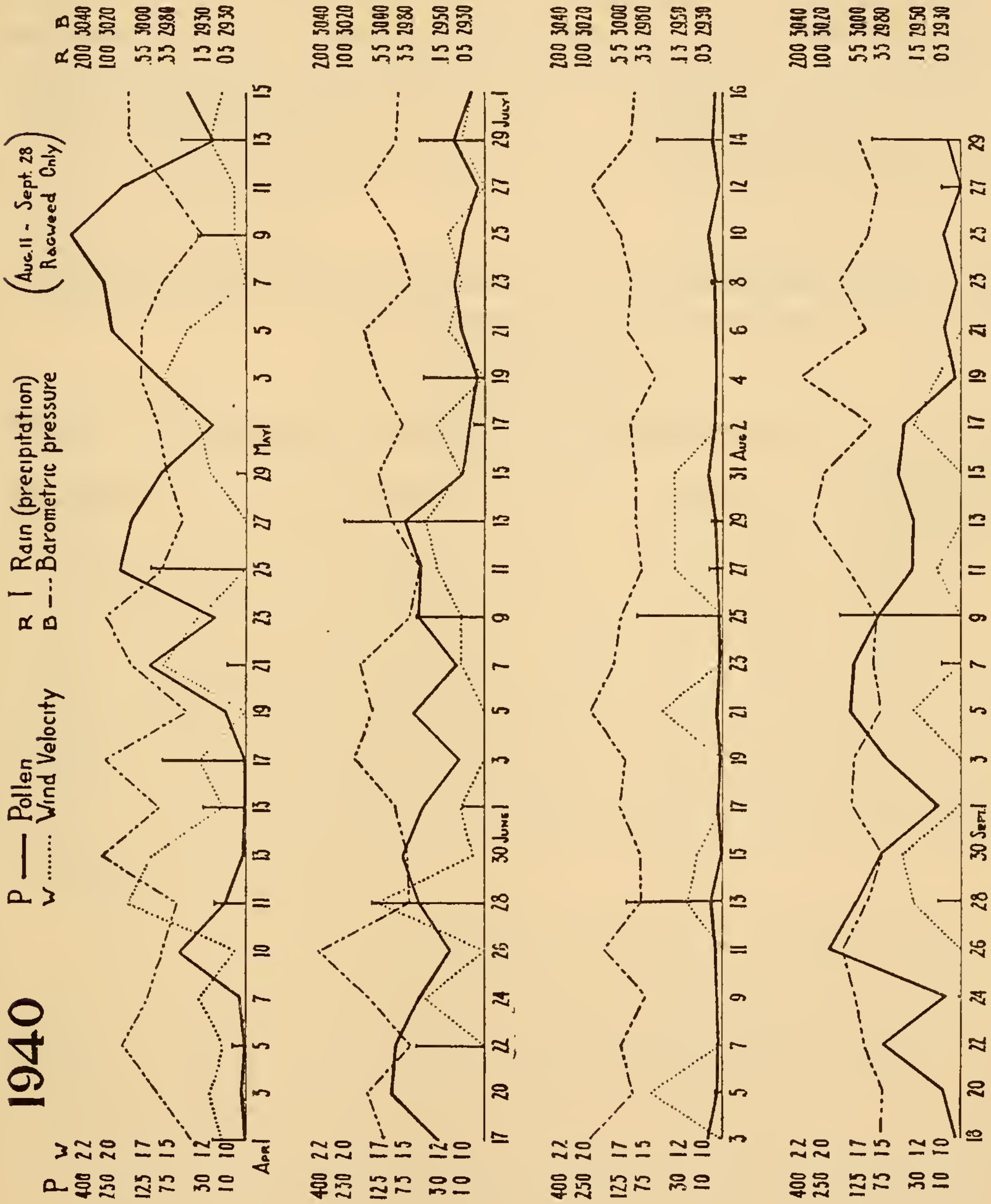

FIG. 1. Correlation of pollen counts, wind velocity, precipitation, and barometric pressure

The chart presented in this issue, therefore, represents the third and covers the significant pollen months for 1939, i.e., April 1, 1939 to

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* From the Allergy Clinic, Department of Medicine, School of Medicine, University of Maryland.

† Received for publication March 6, 1940.

‡ Pollen counts supervised by Selma Rosenberg.

September 29, 1939. In addition to the pollen counts this chart shows also the wind velocity, precipitation, and barometric pressure on the respective days for the purposes of correlation.

COMMENT

There is one notable variation from the average experience revealed by these findings. It is, namely, that the ragweed pollen did not reach a clinically significant concentration until August 22, which is about ten days later than usual. Furthermore, its rise to such concentration was quite abrupt as opposed to the more gradual increase of other years. The clinical findings exactly paralleled the pollen counts; they were late in onset, of shorter duration, and quite intense.

METHOD

The method used was identical with that outlined in the second report (1) and therefore will not be repeated here.

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ONE-HUNDRED-AND-SECOND PROGRAM MEETING

The One-Hundred-and-Second Program Meeting was held on Tuesday, January 16, 1940 in Gordon Wilson Memorial Hall.

Mr. Oskar Baudisch, Research Director of the Saratoga Springs Commission, Saratoga Springs, New York, was the guest speaker and read a paper on "Biological Orientation in Hydrology—Capture, Concentration and Utilization of Trace Elements in Sea Water and in Saratoga Mineral Water."

ONE-HUNDRED-AND-THIRD PROGRAM MEETING

The One-Hundred-and-Third Program Meeting was held on Tuesday, February 13, 1940 in Gordon Wilson Memorial Hall. The following papers were read:

"An Experimental Study of the Effect of the Thyroid on Motility of the Gastro-Intestinal Tract," by Drs. Samuel Morrison and Maurice Feldman, Department of Gastro-Enterology, School of Medicine, University of Maryland.

"Energy Relations in Plant Hormone Transport," by Dr. Herman G. du Buy, University of Maryland, College Park, Md.

Abstracts of these papers follow.

AN EXPERIMENTAL STUDY OF THE EFFECT OF THE THYROID ON MOTILITY OF THE GASTRO-INTESTINAL TRACT* †

SAMUEL MORRISON AND MAURICE FELDMAN

The physiologic mechanism of thyroid action upon the gastro-intestinal tract has not been clearly established in the past. A preliminary study was made to determine the effect of the thyroid and the vagus nerves on gastro-intestinal motility in dogs. These studies yielded the following results:

1. Desiccated thyroid administered to normal dogs showed an acceleration of gastro-intestinal motility.
2. Motility of the stomach is invariably affected by section of the vagus nerves. This procedure causes a six hour retention in almost every instance.
3. Motility of the intestines is not significantly affected by section of the vagus nerves. The immediate effect of vagotomy on the digestive tract is mainly on gastric motility, with very little if any effect upon the intestines.
4. Desiccated thyroid administered to vagotomized dogs revealed acceleration of gastro-intestinal motility, thus demonstrating that thyroid action is independent of the vagus nerves.
5. Removal of the thyroid glands alone had no immediate effect upon gastro-intestinal motility. After a lapse of months, however, it did produce some loss of tone and sluggish motility.
6. Thyroid gland extract given subcutaneously or desiccated thyroid administered orally to the thyroidectomized animals accelerated gastro-intestinal motility.
7. In the combined vagotomized and thyroidectomized dogs, motility studies revealed no immediate changes other than those noted in the vagotomized animals (six hour gastric retention), and indicated that thyroid removal did not influence the changes noted following vagotomy.
8. Desiccated thyroid administered to the animals with combined vagotomy and thyroidectomy revealed a hypermotility of the gastro-intestinal tract, with the return of retention after cessation of thyroid medication.
9. Thyroid medication in adequate dosage produced an acceleration of motility of the intestines as well as the stomach.

ENERGY RELATIONS IN TRANSPORT‡ §

HERMAN G. DU BUY

The translocation of various substances in the plant and possibly in animals (virus diseases transmitted through nerves, etc.) takes place by protoplasmic streaming. Since the protoplasmic streaming is maintained by respiratory processes, respiration primarily regulates the transport.

Thus, fast respiration changes of a few cells had to be measured before this relation could be further established. Heyrovski's polarographic method has been used in devising a microrespirometer (4 μ^3 O_2 uptake from a liquid medium can be measured). It could be demonstrated that a change in respiration was followed by a quantitatively equal change in streaming, and this by an equal change in hormone transport. As a working hypothesis for the understanding of the relation between respiratory processes and streaming, the *rocket hypothesis* of streaming was proposed. Since energy released by respiration is necessarily used for synthesis, movement or heat, the second form of energy must cause the streaming. Using the coacervate model of Kruyt and Bungeberg de Yong it might be conceived that the lipoid-protein coacervate carried a protein-respiratory ferment in a polar position. The release of energy during respiration at this pole (by suddenly changing its surface tension or by actually releasing molecules) might propel the particle. Chance will determine the direction of the streaming. Once streaming has taken place, the particles will remain more or less oriented. Therefore, streaming will in most cases resume its former direction after it has been stopped temporarily.

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* From the Department of Gastro-Enterology, School of Medicine, University of Maryland.

† Read at the meeting of the University of Maryland Biological Society, February 13, 1940.

‡ From the Department of Botany, University of Maryland, College Park, Md.

§ Read at the meeting of the University of Maryland Biological Society, February 13, 1940.

ONE-HUNDRED-AND-FOURTH PROGRAM MEETING

The One-Hundred-and-Fourth Program Meeting was held on Tuesday, March 11, 1940 in Gordon Wilson Memorial Hall. A paper on "Some Biological Problems Depending upon Variation in Populations" was presented by Dr. C. A. Morrell, Senior Pharmacologist of the Laboratory of Hygiene, Department of National Health, Ottawa, Canada.

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The names listed above are officers for the term beginning July 1, 1939 and ending June 30, 1940.

SPRING ACTIVITIES

The Board of Regents, The President and the Board of Directors of the Alumni Association, together with the Acting Dean and Medical Council of the School of Medicine of the University of Maryland, cordially invite you to attend the spring activities.

PROGRAM

May 30, 1940

9:00 A.M.—Registration at the Main University Building.
10:00 A.M.—12 M.—Inspection of University Hospital and School of Medicine.
1:00 P.M.—Luncheon, University Hospital.
2:00 P.M.—Annual Meeting of the Medical Alumni Association, University Hospital.
2:30 P.M.—Dedication of the Frank C. Bressler Research Laboratory.
7:00 P.M.—Annual Banquet, Lord Baltimore Hotel.

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REUNIONS

The following classes have expressed their intention to hold reunions this year:

1890	1905	1920
1895	1910	1925
1900	1915	1930
	1935	

NOTE: The reunions will include the graduates from the College of Physicians and Surgeons, University of Maryland and the Baltimore Medical College. Any other classes desiring to hold reunions will please communicate with the Medical Alumni Office, Lombard and Greene Streets, Baltimore, Maryland, which will be pleased to help in every possible way to make the affair a success.

JUNE 1, 1940

11:00 A.M.—Commencement Exercises, Ritchie Coliseum, College Park, Md.

Secretary of the Medical Alumni Association.

Kindly send . . . . . . ticket . . for the Alumni Banquet of the Medical Alumni Association, University of Maryland, to be held at the Lord Baltimore Hotel on Thursday evening, May 30, 1940, at 7 P.M.

will
I *or* be present for the banquet.
will not

Enclosed please find subscription at $2.50 per ticket.

Name. . . . .
Address. . . . .

(*Detach and mail to:* MEDICAL ALUMNI ASSOCIATION
Lombard and Greene Streets, Baltimore, Md.)

Programs may be obtained at the time of registration.

THE ONE HUNDRED AND THIRTY-THIRD CHARTER DAY CELEBRATION

Alumni, faculty and friends of the University gathered at the Lord Baltimore Hotel on January 20 to celebrate the 133rd Anniversary of Charter Day. His Excellency, Governor Herbert R. O'Conor, was the guest of honor, Dr. Harry C. Byrd, the host. Former Governor Harry W. Nice was toastmaster. The Honorable Clifton A. Woodrum, Virginia Representative in Congress, was the guest speaker. Many prominent national and state officials were present.

Dr. Byrd expressed his gratitude to the alumni and faculty for the part they are taking in the progress of our Alma Mater. Governor O'Conor assured him of the cooperation of the State Administration. "American Frontiers" was the topic of the guest speaker, the Honorable Clifton A. Woodrum.

Special observance was accorded the Centenary Celebration of the Dental School, which is the oldest in the world.

ITEMS

At the meeting of the Maryland Academy of Medicine and Surgery held on January 16, 1940 at the Belvedere Hotel, Dr. J. Mason Hundley, Jr., Professor of Gynecology, discussed "Early Diagnosis of Cancer of the Cervix." The lecture was illustrated by lantern slides.

Dr. Arthur M. Shipley, Professor of Surgery, was a speaker on the same program. His topic was "Myositis Ossificans Traumatica."

Dr. Thomas S. Saunders (U. of Md. 1932) was recently certified as a specialist by examination of the American Board of Dermatology and Syphilology. Since February, 1937 Dr. Saunders has been practicing in Portland, Oregon and is Clinical Associate in Dermatology and Syphilology at the University of Oregon Medical School.

Dr. E. N. Boccanegra Lopez (U. of Md. 1916), lecturer in dermatology at the New York Polyclinic Medical School, was made a Diplomate at the recent meeting of the American Board of Dermatology and Syphilology.

Dr. Henry F. Ullrich, Associate in Surgery and Orthopedic Surgery, participated in the program of the meeting of the Maryland Academy of Medicine and Surgery which was held on December 19, 1939 at the Belvedere Hotel. His topic was "The Treatment of Scoliosis." On the same program Dr. William S. Love, Jr., Associate Professor of Medicine and Instructor in Pathology, spoke on "Primary Cor Pulmonalis (Right Heart Disease)."

OBITUARIES

GEORGE C. McELFATRICK, M.D.

Dr. George C. McElfatrick died suddenly on March 20, 1940, while attending a meeting of the Executive Committee of the Delaware Hospital in Wilmington, Delaware.

Dr. McElfatrick was born on July 18, 1881 at Bellaghy, County Derry, Ireland. He was graduated at the Baltimore Medical College in the class of 1910. Dr. McElfatrick was chief of the Departments of Roentgenology in the Delaware and Delaware State Hospitals in Wilmington, Delaware, and held membership in the New Castle Medical Society, the Philadelphia Roentgen Ray Society, the Radiological Society of North America, the Delaware State Medical Society and the American Medical Association. He was a diplomate of the American Board of Radiology and a fellow of the American College of Radiology. In 1930 he was elected President of the Delaware State Medical Society.

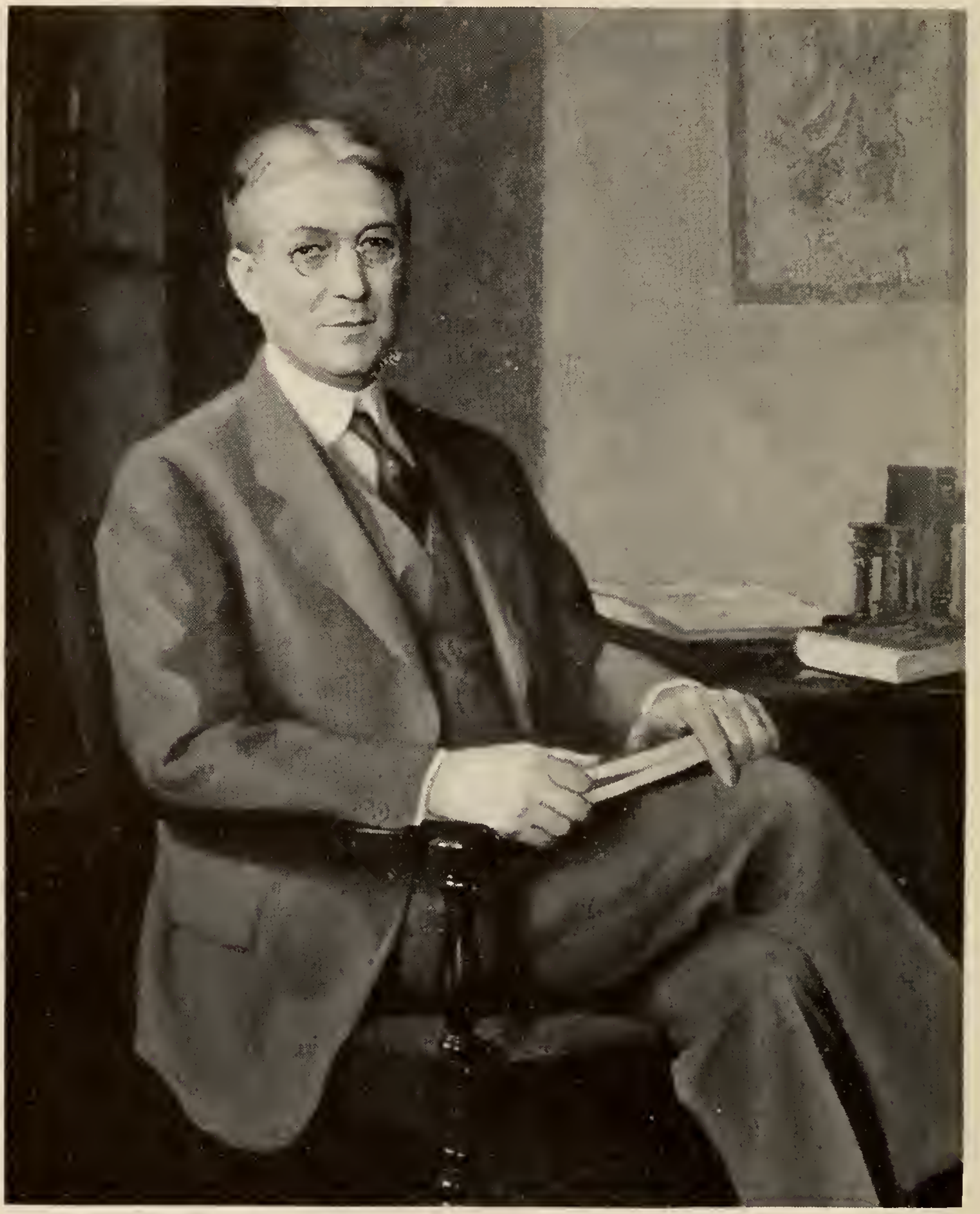
Dr. McElfatrick achieved success in his chosen profession through diligent, conscientious work. He possessed a keen sense of humor and spirit of optimism and will be greatly missed by his colleagues and by those of us who were fortunate enough to know him as a sincere friend.

ALEXIUS McGLANNAN, A.M., M.D., LL.D.

Dr. Alexius McGlannan died at his home, 115 West Franklin Street, Baltimore, on Sunday, February 25, 1940 after an illness of considerable duration. Dr. McGlannan was born in Baltimore on July 24, 1872, his parents being Alexius W. McGlannan and Agnes Veronica Gallagher McGlannan. His preliminary education was received at Calvert Hall College and he was graduated in medicine by the College of Physicians and Surgeons, now combined with the University of Maryland, in 1895, and soon became actively engaged in teaching in this institution.

At first he seemed inclined to medical subjects with a particular interest in chemistry, but soon decided that surgery was to be his life's work and became connected with this department, quickly working his way upward. He had the conception, more universally stressed by many later, that the study of pathology is an important foundation to good surgery, and became rather closely associated with the late Dr. Joseph C. Bloodgood at his laboratory at the Johns Hopkins Hospital and in his clinical work at St. Agnes Hospital.

Dr. McGlannan's great energy, his fidelity to his work and an unusual memory soon bore fruit in rapid advancement in his chosen specialty. By 1915 he had become Professor of Clinical Surgery and Surgical Pathology of the College of Physicians and Surgeons. In 1930 he was made Professor of Surgery at the School of Medicine, University of Maryland. In 1926 he was made Chief Surgeon of



Alexius McGlannan, A.M., M.D., LL.D.

Mercy Hospital, where most of his work had been done and where he advanced step by step.

These positions entailed so much work that he eventually declined to operate except in the hospital with which he was so closely allied and for which he did so much, though he was on the staffs of several others. In 1933 his health became impaired and necessitated curtailment of his activities, but by this time he had done a prodigious amount of work and achieved many honors. In addition to the positions already mentioned, he had been honored by numerous offices in the Medical and Chirurgical Faculty of Maryland, doing almost unlimited work on its committees, and was elected its President in 1929.

In addition to the City and State Societies and the American Medical Association, he was a member of the American College of Surgeons, Southern Surgical Association and American Surgical Association, and contributed many papers to these societies and to medical journals. In fact, it might be said that during his active years, besides the operative work of a large clinic, much teaching and caring for a large private practice, he wrote a great deal. One sees his articles in systems of surgery and medicine and his name in many bibliographies. The number of his published articles totals nearly one hundred.

Apparently Dr. McGlannan felt that "opportunity is kind but only to the industrious." He had none of the usual avocations, such as golf, hunting or fishing, though he did some boating on Saranac Lake where he spent the summers at his elaborate camp. He did not drink, smoke or play cards. General reading and music were his recreations. He never ceased studying, even when he no longer operated.

Dr. McGlannan's reading was of a wide range with surgery as the central theme but he seemed to gather information easily and retain it permanently about all manner of subjects. This naturally added to his value as a consultant in diagnosis and treatment, and enabled him to surpassingly well avoid being the type of surgeon who is only an operator.

Dr. McGlannan's first wife, who was Miss Anna Maria Crean, died in 1902. They had one son, Alexius McGlannan, III. In 1907 he married Dr. Sallie Porter Law, who was graduated in medicine at the Johns Hopkins Medical School that year but never practiced. For thirty-three years she was a devoted helpmate, doing her utmost to shield her husband from stress and strain and, after the break in his health came, giving him every attention, not leaving the house for months, and during the last days doing twenty-four hour nursing. Mrs. McGlannan is a gifted artist and illustrated Dr. McGlannan's surgical papers. There were no children by this marriage. He is survived by Mrs. McGlannan, his son by his first marriage, and nieces and nephews.

One could pay tribute to Dr. McGlannan's ability, his industry, his accomplishments in surgery, or his devotion to the Roman Catholic religion and his exemplary life, but to many colleagues, house officers, students and countless patients, his outstanding characteristics were friendliness and kindness and, above all, charitableness. He lent strength to the quotation "charity suffereth long and is kind; charity envieth not; charity vaunteth not itself, is not puffed up." He followed the admonition

> "So to live; that one may give
> A helping hand,—a cheering smile
> To those who find their journey sad
> For yet a while."

WALTER D. WISE.

Barnes, Benjamin Franklin, Elm City, N. C.; class of 1902; aged 62; died, December 31, 1939, of cerebral hemorrhage.

Broadwater, Norman Irving, Oakland, Md.; class of 1909; aged 57; died, January 3, 1940, in the Memorial Hospital at Cumberland of cerebral hemorrhage.

Cobey, James C., Frostburg, Md.; P. & S., class of 1896; died, February 1, 1940.

Crane, James Douglas, Ishpeming, Mich.; P. & S., class of 1914; served during the World War; aged 47; died, December 18, 1939, in the Columbia Hospital, Milwaukee, of coronary disease and chronic hypertension.

Earle, Claude E., Anderson, S. C.; P. & S., class of 1886; aged 79; died, December 23, 1939, in the Anderson County Hospital, of carcinoma of the prostate.

Ferry, Bernard Joseph, Baltimore, Md.; class of 1916; aged 53, died, December 13, 1939, of myeloid leukemia.

Finley, Francis Howard, Finleyville, Pa.; P. & S., class of 1895; aged 68; died, December 25, 1939, of bronchopneumonia.

Hammond, George Abner, Dothan, Ala.; B.M.C., class of 1884; aged 80; died, January 4, 1940, of chronic myocarditis.

Hanes, John Lewis, Pine Hall, N. C.; class of 1902; aged 60; died, January 16, 1940.

Harrell, Julian Paul, Brunswick, Ga.; class of 1906; aged 58; died, December 27, 1939, in Thomaston, of injuries received in an automobile accident.

Johnson, George Lewis, Morristown, N. J.; B.M.C., class of 1901; served during the World War; aged 67; died, December 12, 1939, of cerebral arteriosclerosis.

Johnson, Thomas Brown, Indianapolis, Ind.; class of 1906; served during the World War; aged 57; died, January 6, 1940, of coronary thrombosis.

Kirksey, Robert, Pickens, S. C.; P. & S., class of 1891; aged 72; died, December 29, 1939, of pneumonia.

Knight, Herbert Wilcox, Bostic, N. C.; B.M.C., class of 1903; aged 66; died, November 4, 1939, in the Rutherford (N. C.) Hospital, of chronic myocarditis.

McGuire, G. T., Wyoming, Pa.; B.M.C., class of 1898; aged 72; died, November 19, 1939, of cerebral hemorrhage.

Myers, John W., Glen Dale, W. Va.; P. & S., class of 1893; aged 68; died, December 19, 1939, of pneumonia.

Nuckols, C. Lunsford, Baltimore, Md.; B.M.C., class of 1893; aged 68; died, November 10, 1939, of lobar pneumonia.

Patton, David H. R., Chicago, Ill.; B.M.C., class of 1896; aged 73; died, February 28, 1940.

Phillips, George Marquet, Eau Gallie, Fla.; P. & S., class of 1887; aged 77; died, December 25, 1939, in the Riverside Hospital, Jacksonville, of cerebral hemorrhage.

Pinsky, Meyer Mordecai, Camden, N. J.; class of 1925; aged 38; died, October 6, 1939, of coronary thrombosis.

Robins, William Littleton, Washington, D. C.; class of 1890; aged 72; died, January 29, 1940.

Rogers, Francis Owington, Little Rock, Ark.; class of 1901; aged 63; died, November 8, 1939, in the Lynnhurst Sanatorium, Oakville, Tenn.

Savage, Rupert L., Rocky Mount, N. C.; class of 1897; aged 65; died, November 15, 1939, of coronary thrombosis.

Shields, Thomas K., Wheeling, W. Va.; class of 1890; aged 74; died, October 7, 1939, of chronic nephritis, myocarditis and arteriosclerosis, hypertension and cerebral hemorrhage.

Thieme, Gustav Charles, Baltimore, Md.; P. & S., class of 1896; aged 68; died, November 5, 1939, of carcinoma of the rectum.

Walke, Frank Hicks, Shreveport, La.; B.M.C., class of 1912; served during the World War; aged 53; died, December 21, 1939.

Wetzel, Edward T., West Union, W. Va.; B.M.C., class of 1903; aged 67; died, November 4, 1939, of cerebral hemorrhage.

Wilkinson, Edward L., Jacksonville, Fla.; P. & S., class of 1885; aged 79; died, November 6, 1939, in St. Luke's Hospital, of mesenteric thrombosis.

Zion, Elkanah, Knoxville, Tenn.; class of 1888; aged 76; died, December 6, 1939, of arteriosclerosis.

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The Frank C. Bressler Research Laboratory

BULLETIN
OF THE
SCHOOL *of* MEDICINE
UNIVERSITY OF MARYLAND

Announcement
for the
One Hundred Thirty-Fourth Annual Session
1940-1941

VOLUME 24, NO. 5
MAY, 1940

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MARCH

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22	23	24	25	26	27	28
29	30	31	....	....	....	....
....	....	....	....	....	....	....

APRIL

S	M	T	W	T	F	S
....	....	....	1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30	....	....

MAY

S	M	T	W	T	F	S
....	....	....	....	....	1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30
31	....	....	....	....	....	....

JUNE

S	M	T	W	T	F	S
....	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	....	....	....	....

CALENDAR 1940–41, SCHOOL OF MEDICINE

1940		
September 19–21		Conditioned Examinations

FIRST SEMESTER

September 24	Tuesday	*Registration, freshmen and sophomores
September 25	Wednesday	*Registration, all other students
September 26	Thursday	Instruction begins
November 20	Wednesday	Instruction suspended at 5:00 P.M.
		Thanksgiving Holidays
November 25	Monday	Instruction resumed
December 21	Saturday	Instruction suspended at 2:00 P.M.
1941		Christmas Holidays
January 6	Monday	Instruction resumed
January 25	Saturday	Instruction suspended at 2:00 P.M.
January 27 to February 1	Monday to Saturday	Examinations, all classes, and *Registration for second semester
February 1	Saturday	First semester completed at 12:30 P.M.

SECOND SEMESTER

February 3	Monday	Instruction resumed
February 21	Friday	Instruction suspended at 5:00 P.M.
February 22	Saturday	Holiday—Washington's Birthday
February 24	Monday	Instruction resumed
April 9	Wednesday	Instruction suspended at 5:00 P.M.
		Easter Holidays
April 16	Wednesday	Instruction resumed
May 17	Saturday	Senior and junior instruction ends at 2:00 P.M.
May 19	Monday	Senior and junior examinations begin
May 24	Saturday	Senior examinations completed Sophomore and freshman instruction ends at 12:00 M.
May 26	Monday	Junior examinations continue Sophomore and freshman examinations begin
May 31	Saturday	Announcement of graduates All examinations completed Second semester completed at 12:30 P.M.
June 7	Saturday	Commencement at 11:00 A.M.

PARTIAL CALENDAR FOR 1941–1942

1941		
September 18–20		Conditioned Examinations
September 23	Tuesday	*Registration, freshmen and sophomores
September 24	Wednesday	*Registration, all other students
September 25	Thursday	Instruction begins

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* A student who fails to register prior to or within the day or days specified will be called upon to pay a late registration fee of five dollars ($5.00). The last day of registration with fee added to regular charges is Saturday of the week in which registration begins.

The offices of the registrar and comptroller are open daily, not including Saturday, from 9:00 a.m. to 5:00 p.m., and Saturday from 9:00 a.m. to 12:30 p.m.

Early registration is encouraged.

ORGANIZATION

THE UNIVERSITY OF MARYLAND

Harry Clifton Byrd, B.S., LL.D., D.Sc., *President and Executive Officer*

BOARD OF REGENTS

	Term expires
Dr. W. W. Skinner, *Chairman* Kensington, Montgomery County	1945
Mrs. John L. Whitehurst, *Secretary* 4101 Greenway, Baltimore	1947
J. Milton Patterson, Esq., *Treasurer* 1015 Argonne Drive, Baltimore	1944
Hon. W. Calvin Chesnut Roland Park, Baltimore	1942
Hon. William P. Cole, Jr Towson, Baltimore County	1940
Henry Holzapfel, Jr., Esq Hagerstown, Washington County	1943
Harry H. Nuttle, Esq Denton, Caroline County	1941
John E. Semmes, Esq 100 W. University Parkway, Baltimore	1942
Hon. Rowland K. Adams 1808 Fairbank Road, Baltimore	1948

The control of the University of Maryland is vested in a Board of nine Regents, appointed by the Governor and confirmed by the Senate for terms of nine years each. The general administration of the University is vested in the President. The University Council is an advisory body, composed of the President, the Director of the Agricultural Experiment Station, the Director of the Extension Service, and the Deans. The University Council acts upon all matters having relation to the University as a whole, or to co-operative work between the constituent groups. Each school has its own Faculty Council, composed of the Dean and members of its faculty; each Faculty Council controls the internal affairs of the group it represents.

The University has the following educational organizations:

At Baltimore

The School of Dentistry
The School of Law
The School of Medicine
The School of Nursing
The School of Pharmacy

At College Park

The College of Agriculture
The College of Arts and Sciences
The College of Commerce
The College of Education
The College of Engineering
The College of Home Economics
The Graduate School
The Department of Military Science and Tactics
The Department of Physical Education and Recreation
The Summer School

THE UNIVERSITY SENATE
(Baltimore Schools)

H. C. Byrd, B.S., LL.D., D.Sc.	President of the University
H. Boyd Wylie, M.D.	Acting Dean of the School of Medicine
Roger Howell, Ph.D., LL.B.	Dean of the School of Law
Andrew G. DuMez, M.S., Ph.D.	Dean of the School of Pharmacy
J. Ben Robinson, D.D.S., F.A.C.D.	Dean of the School of Dentistry
L. B. Broughton, Ph.D.	Dean of the College of Arts and Sciences
[1]Arthur J. Lomas, M.D., C.M., D.P.H	Superintendent of the Hospital
Alma H. Preinkert, M.A.	Registrar
W. M. Hillegeist	Director of Admissions

FACULTY OF PHYSIC

EMERTI

J. Frank Crouch, M.D.	Professor of Clinical Ophthalmology and Otology, Emeritus
Harry Friedenwald, A.B., M.D., D.H.L., D.Sc.	Professor of Ophthalmology, Emeritus
William S. Gardner, M.D.	Professor of Gynecology, Emeritus
Julius Friedenwald, A.M., M.D.	Professor of Gastro-Enterology, Emeritus
§Alexius McGlannan, A.M., M.D., LL.D.	Professor of Surgery, Emeritus
J. M. H. Rowland, M.D., Sc.D., LL.D.	Professor of Obstetrics, Emeritus; Dean, Emeritus

MEDICAL COUNCIL

H. Boyd Wylie, *Acting Dean*

Arthur M. Shipley
Hugh R. Spencer
Carl L. Davis
Maurice C. Pincoffs
Frank W. Hachtel
Eduard Uhlenhuth

Clyde A. Clapp
John C. Krantz, Jr.
Walter D. Wise
J. Mason Hundley, Jr.
William R. Amberson
Louis H. Douglass

PROFESSORS

Arthur M. Shipley, M.D., Sc.D., Professor of Surgery.
Hugh R. Spencer, M.D., Professor of Pathology.
H. Boyd Wylie, M.D., Professor of Biological Chemistry and Acting Dean.
Carl L. Davis, M.D., Professor of Anatomy.
Maurice C. Pincoffs, B.S., M.D., Professor of Medicine.
Frank W. Hachtel, M.D., Professor of Bacteriology.
Clyde A. Clapp, M.D., Professor of Ophthalmology.
John C. Krantz, Jr., Ph.D., Professor of Pharmacology.
Eduard Uhlenhuth, Ph.D., Professor of Anatomy.
J. Mason Hundley, Jr., M.A., M.D., Professor of Gynecology.
Walter D. Wise, M.D., Professor of Surgery.
William R. Amberson, Ph.D., Professor of Physiology.
Louis H. Douglass, M.D., Professor of Obstetrics.

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[1] Retired December 31, 1939.

§Deceased February 25, 1940.

Joseph E. Gichner, M.D., Professor of Clinical Medicine and Physical Therapeutics.
Irving J. Spear, M.D., Professor of Neurology.
Charles F. Blake, A.M., M.D., Professor of Diseases of the Rectum and Colon.
S. Griffith Davis, M.S., M.D., Professor of Anaesthesia.
G. Carroll Lockard, M.D., Professor of Clinical Medicine.
Harvey G. Beck, M.D., Sc.D., Professor of Clinical Medicine.
Albertus Cotton, A.M., M.D., Professor of Orthopaedic Surgery and Roentgenology.
Andrew C. Gillis, A.M., M.D., LL.D., Professor of Neurology.
Henry J. Walton, M.D., Professor of Roentgenology.
Ross McC. Chapman, M.D., Professor of Psychiatry.
Edgar B. Friedenwald, M.D., Professor of Clinical Pediatrics.
Edward A. Looper, M.D., D.Oph., Professor of Diseases of the Nose and Throat.
J. W. Downey, M.D., Professor of Otology.
Harry M. Stein, M.D., Professor of Clinical Medicine.
W. H. Toulson, A.B., M.Sc., M.D., Professor of Genito-Urinary Surgery.
Charles Bagley, Jr., M.A., M.D., Professor of Neurological Surgery.
Allen Fiske Voshell, A.B., M.D., Professor of Orthopaedic Surgery.
Page Edmunds, M.D., Professor of Traumatic Surgery.
Harry M. Robinson, M.D., Professor of Dermatology.
Elliott H. Hutchins, A.M., M.D., Professor of Clinical Surgery.
Charles Reid Edwards, M.D., Professor of Clinical Surgery.
F. L. Jennings, M.D., Professor of Clinical Surgery.
Huntington Williams, M.D., Dr.P.H., Professor of Hygiene and Public Health.
§Robert P. Bay, M.D., Professor of Oral Surgery.
C. Loring Joslin, M.D., Professor of Pediatrics.
Brice M. Dorsey, D.D.S., Professor of Exodontia.
J. Dawson Reeder, M.D., Professor of Diseases of the Rectum and Colon.
Thomas P. Sprunt, A.B., M.D., Professor of Clinical Medicine.
Thomas B. Aycock, B.S., M.D., Clinical Professor of Surgery.
Compton Riely, M.D., Clinical Professor of Orthopaedic Surgery.
W. S. Smith, M.D., Clinical Professor of Gynecology.
M. Randolph Kahn, M.D., Clinical Professor of Ophthalmology.
Waitman F. Zinn, M.D., Clinical Professor of Diseases of the Nose and Throat.
A. J. Gillis, M.D., Clinical Professor of Genito-Urinary Surgery.
Theodore H. Morrison, M.D., Clinical Professor of Gastro-Enterology.
Abram S. Samuels, A.B., M.D., Clinical Professor of Gynecology.
H. K. Fleck, M.D., Clinical Professor of Ophthalmology.

ASSOCIATE PROFESSORS

R. W. Locher, M.D., Associate Professor of Clinical Surgery.
H. R. Peters, A.B., M.D., Associate Professor of Medicine.
T. Nelson Carey, M.D., Associate Professor of Medicine and Physician in Charge of Medical Care of Students.
Howard J. Maldeis, M.D., Associate Professor of Medical Jurisprudence and Associate in Pathology.
G. M. Settle, A.B., M.D., Associate Professor of Neurology and Clinical Medicine.
Thomas R. Chambers, A.B., M.D., Associate Professor of Surgery.
William H. Smith, M.D., Associate Professor of Clinical Medicine.
Paul W. Clough, B.S., M.D., Associate Professor of Medicine.

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§ Deceased January 1, 1940.

Sydney R. Miller, B.S., M.D., Associate Professor of Medicine.
J. McFarland Bergland, B.S. M.D., Associate Professor of Obstetrics.
Walter A. Baetjer, A.B., M.D.. Associate Professor of Medicine.
Edward S. Johnson, M.D., Associate Professor of Surgery.
Emil Novak, A.B., M.D., D.Sc., Associate Professor of Obstetrics.
Ralph P. Truitt, M.D., Associate Professor of Psychiatry.
D. J. Pessagno, A.B., M.D., Associate Professor of Surgery.
Moses Gellman, B.S., M.D., Associate Professor of Orthopaedic Surgery.
Harry L. Rogers, M.D., Associate Professor of Orthopaedic Surgery.
O. G. Harne, Associate Professor of Histology.
Charles A. Reifschneider, M.D., Associate Professor of Traumatic Surgery and Oral Surgery.
Monte Edwards, M.D., Associate Professor of Surgery and of Diseases of the Rectum and Colon.
[1]Frank H. J. Figge, Ph.D., Associate Professor of Gross Anatomy.
Wm. S. Love, Jr., A.B., M.D., Associate Professor of Medicine and Instructor in Pathology.
Raymond Hussey, M.A., M.D., Associate Professor of Medicine.
L. A. M. Krause, M.D., Associate Professor of Medicine.
N. Clyde Marvel, A.B., M.D., Associate Professor of Surgery.
T. Campbell Goodwin, M.S., M.D., Associate Professor of Pediatrics.
Carl Dame Clarke, M.A., Associate Professor of Art as Applied to Medicine.
Emil G. Schmidt, Ph.D., LL.B., Associate Professor of Biological Chemistry.
Lawrence F. Woolley, M.D., Associate Professor of Psychiatry.
Richard G. Coblentz, M.A., M.D., Associate Professor of Neurological Surgery.
Grant E. Ward, A.B., M.D., Associate Professor of Surgery.
Dietrich Conrad Smith, Ph.D., Associate Professor of Physiology.
Franklin B. Anderson, M.D., Associate Professor of Diseases of the Nose and Throat and Otology.
Leon Freedom, M.D., Associate Professor of Neurology and Instructor in Pathology.
§Milford Levy, M.D., Associate Professor of Neurology.
James G. McAlpine, Ph.D., Associate Professor of Bacteriology.
C. Jelleff Carr, Ph.D., Associate Professor of Pharmacology.
Cyrus F. Horine, M.D., Associate Professor of Surgery.
Kenneth D. Legge, M.D., Associate Professor of Genito-Urinary Surgery.

ASSISTANT PROFESSORS

George McLean, M.D., Assistant Professor of Medicine.
Maurice Feldman, M.D., Assistant Professor of Gastro-Enterology.
Robert B. Wright, B.S., M.D., Assistant Professor of Pathology.
John H. Traband, M.D., Assistant Professor of Pediatrics.
Albert Jaffe, M.D., Assistant Professor of Pediatrics.
C. Gardner Warner, A.B., M.D., Assistant Professor of Pathology.
Harry Goldsmith, M.D., Assistant Professor of Psychiatry.
H. W. Newell, M.D., Assistant Professor of Psychiatry.
Zachariah Morgan, M.D., Assistant Professor of Gastro-Enterology.
Samuel Morrison, M.D., Assistant Professor of Gastro-Enterology.
Harry M. Murdock, B.S., M.D., Assistant Professor of Psychiatry.
A. H. Finkelstein, M.D., Assistant Professor of Pediatrics.

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[1] On leave, 1940–1941.

§ Deceased, October 10, 1939.

John E. Legge, M.D., Assistant Professor of Medicine.
Thomas C. Wolff, Litt.B., M.D., C.M., Assistant Professor of Medicine.
Frederick B. Smith, M.D., Assistant Professor of Pediatrics.
J. G. M. Reese, M.D., Assistant Professor of Obstetrics.
M. Alexander Novey, A.B., M.D., Assistant Professor of Obstetrics.
Thomas K. Galvin, M.D., Assistant Professor of Gynecology.
Leo Brady, A.B., M.D., Assistant Professor of Gynecology.
R. G. Willse, M.D., Assistant Professor of Gynecology.
George A. Strauss., Jr, M.D., Assistant Professor of Gynecology.
Robert H. Oster, Ph.D., Assistant Professor of Physiology.
H. M. Bubert, M.D., Assistant Professor of Medicine.
Walter C. Merkel, A.B., M.D., Assistant Professor of Pathology.
John F. Lutz, A.B., M.D., Assistant Professor of Histology.
James C. Lipsett, Ph.D., Assistant Professor of Gross Anatomy.
Benjamin Pushkin, M.D., Assistant Professor of Neurology.
William Ellsworth Evans, Ph.D., Assistant Professor of Pharmacology.
H. F. Bongardt, M.D., Assistant Professor of Surgery.
J. Edmund Bradley, B.S., M.D., Assistant Professor of Pediatrics.
Walter L. Kilby, M.D., Assistant Professor of Roentgenology.
George H. Yeager, B.S., M.D., Assistant Professor of Surgery.
Isadore A. Siegel, A.B., M.D., Assistant Professor of Obstetrics.
William H. F. Warthen, A.B., M.D., Assistant Professor of Hygiene and Public Health.
*Francis Ellis, A.B., M.D., Assistant Professor of Dermatology.

LECTURERS

Jonas Friedenwald, M.A., M.D., Lecturer in Ophthalmic Pathology.
E. B. Freeman, B.S., M.D., Lecturer in Medicine.
Charles R. Goldsborough, M.A., M.D., Lecturer in Medicine.
W. N. Bispham, Col., M.C., U. S. A. (ret.), Lecturer in Medicine
†Leslie B. Hohman, M.D., Lecturer in Psychiatry.

ASSOCIATES

John R. Abercrombie, A.B., M.D., Associate in Dermatology.
Joseph I. Kemler, M.D., Associate in Ophthalmology.
Frank N. Ogden, M.D., Associate in Biological Chemistry.
Edward P. Smith, M.D., Associate in Gynecology.
Austin H. Wood, M.D., Associate in Genito-Urinary Surgery.
Albert E. Goldstein, M.D., Associate in Pathology.
Samuel S. Glick, M.D., Associate in Pediatrics.
William J. Todd, M.D., Associate in Pediatrics.
Clewell Howell, B.S., M.D., Associate in Pediatrics.
Joseph Sindler, M.D., Associate in Gastro-Enterology.
I. O. Ridgley, M.S., M.D., Associate in Surgery.
R. D. West, M.D., Associate in Ophthalmology.
Eugene L. Flippin, M.D., Associate in Roentgenology.
J. S. Eastland, A.B., M.D., Associate in Medicine.
L. J. Millan, M.D., Associate in Genito-Urinary Surgery.
W. Raymond McKenzie, M.D., Associate in Diseases of the Nose and Throat.
E. P. H. Harrison, A.B., M.D., Associate in Obstetrics.

L. K. Fargo, M.D., Associate in Genito-Urinary Surgery.
Henry F. Graff, A.B., M.D., Associate in Ophthalmology.
Harold M. Goodman, A.B., M.D., Associate in Dermatology.
Wetherbee Fort, M.D., Associate in Medicine.
F. Stratner Orem, M.D., Associate in Pediatrics.
Frank J. Geraghty, A.B., M.D., Associate in Medicine.
L. P. Gundry, A.B., M.D., Associate in Medicine.
John F. Hogan, M.D., Associate in Genito-Urinary Surgery.
Thomas R. O'Rourk, M.D., Associate in Diseases of the Nose and Throat and Otology and Assistant in Ophthalmology.
Kenneth B. Boyd, M.D., Associate in Gynecology and Assistant in Obstetrics.
John T. Hibbits, M.D., Associate in Gynecology.
E. H. Tonolla, M.D., Associate in Medicine.
C. W. Peake, M.D., Associate in Surgery.
W. A. H. Councill, M.D., Associate in Genito-Urinary Surgery.
Z. Vance Hooper, M.D., Associate in Gastro-Enterology.
Frank B. Kindell, A.B., M.D., Associate in Pathology.
William M. Seabold, A.B., M.D., Associate in Pediatrics.
H. Alvan Jones, M.D., Associate in Orthopaedic Surgery.
Henry F. Ullrich, M.D., Sc.D., Associate in Surgery and Orthopaedic Surgery.
Benjamin S. Rich, A.B., M.D., Associate in Otology.
John E. Savage, B.S., M.D., Associate in Obstetrics and Assistant in Pathology.
*Joseph C. Bernstein, M.D., Associate in Dermatology.
*Thomas J. Coonan, A.B., M.D., Associate in Pediatrics.
*†Eugene E. Covington, M.D., *Associate in Gross Anatomy and †Instructor in Surgery.

INSTRUCTORS

Henry F. Buettner, M.D., Instructor in Bacteriology.
J. A. F. Pfeiffer, Ph.D., M.D., Instructor in Bacteriology.
M. S. Koppelman, M.D., Instructor in Gastro-Enterology.
Myron G. Tull, A.B., M.D., Instructor in Hygiene and Public Health.
William R. Johnson, M.D., Instructor in Surgery.
E. M. Hanrahan, A.B., M.D., Instructor in Surgery.
Ruth Musser, A.B., M.S., Instructor in Pharmacology.
Benjamin Abeshouse, Ph.B., M.D., Instructor in Pathology.
Joseph Pokorney, M.D., Instructor in Histology.
S. Demarco, M.D., Instructor in Surgery.
Karl J. Steinmueller, A.B., M.D., Instructor in Surgery.
Samuel T. Helms, B.S., M.D., Instructor in Medicine and Genito-Urinary Surgery.
F. A. Holden, M.D., Instructor in Ophthalmology.
Martin J. Hanna, M.D., Instructor in Histology.
Earl L. Chambers, M.D., Instructor in Medicine.
M. Paul Byerly, M.D., Instructor in Pediatrics.
G. Bowers Mansdorfer, B.S., M.D., Instructor in Pediatrics.
William E. Hahn, D.D.S., Instructor in Exodontia.
Luther E. Little, M.D., Instructor in Surgery.
J. Frank Hewitt, A.B., M.D., Instructor in Surgery.
David Tenner, M.D., Instructor in Medicine.
R. B. Mitchell, Jr., B.S., M.D., Instructor in Medicine.

Samuel Legum, A.B., M.D., Instructor in Medicine.
Ernest Levi, M.D., Instructor in Gastro-Enterology.
Ernest S. Edlow, A.B., M.D., Instructor in Gynecology.
Milton S. Sacks, B.S., M.D., Instructor in Pathology.
Thomas S. Bowyer, A.B., M.D., Instructor in Gynecology.
Joseph V. Castagna, M.D., Instructor in Gynecology.
Sol Smith, A.B., M.D., Instructor in Medicine.
M. S. Shiling, A.B., M.D., Sc.D., Instructor in Medicine.
Robert W. Garis, A.B., M.D., Instructor in Medicine.
Harry V. Langeluttig, A.B., M.D., Instructor in Medicine.
Simon H. Brager, M.D., Instructor in Surgery.
James W. Nelson, M.D., Instructor in Surgery.
I. Ridgeway Trimble, M.D., Instructor in Surgery.
Richard T. Shackelford, A.B., M.D., Instructor in Surgery.
Raymond F. Helfrich, A.B., M.D., Instructor in Surgery.
Harry S. Shelley, B.S., M.D., Instructor in Genito-Urinary Surgery and Gross Anatomy.
Harry C. Hull, M.D., Instructor in Surgery and Assistant in Pathology.
Philip F. Lerner, A.B., M.D., Instructor in Neurology.
H. Edmund Levin, B.S., M.D., Instructor in Medicine and Bacteriology.
Conrad B. Acton, B.S., M.D., Instructor in Medicine and Assistant in Pathology.
Robert A. Reiter, A.B., M.D., Instructor in Medicine.
Harry M. Robinson, Jr., B.S., M.D., Instructor in Dermatology and Assistant in Medicine.
W. Grafton Hersperger, A.B., M.D., Instructor in Medicine.
Meyer W. Jacobson, M.D., Instructor in Medicine.
Hugh G. Whitehead, M.D., Instructor in Medicine.
F. Edwin Knowles, Jr., M.D., Instructor in Ophthalmology.
Philip D. Flynn, M.D., Instructor in Medicine.
Otto C. Brantigan, B.S., M.D., Instructor in Gross Anatomy and Assistant in Surgery.
Albert J. Shochat, B.S., M.D., Instructor in Gastro-Enterology.
Thomas A. Christensen, B.S., M.D., Instructor in Pediatrics.
Jerome Fineman, M.D., Instructor in Pediatrics.
Bernhard Badt, M.D., Instructor in Neurology.
William L. Fearing, M.D., Instructor in Neurology.
Frank K. Morris, A.B., M.D., Instructor in Gynecology and Obstetrics.
B. Matthew Debuskey, M.D., Instructor in Pediatrics.
A. Russell Anderson, M.D., Instructor in Psychiatry.
D. James Greiner, B.S., M.D., Instructor in Pathology.
Francis W. Gillis, M.D., Instructor in Genito-Urinary Surgery.
James C. Owings, M.D., Instructor in Diseases of the Rectum and Colon.
*Edward S. Kallins, B.S., M.D., Instructor in Medicine.
*John C. Dumler, B.S., M.D., Instructor in Gynecology.
*Beverley C. Compton, A.B., M.D., Instructor in Gynecology.
*W. Allen Deckert, A.B., M.D., Instructor in *Gynecology and Assistant in Surgery and Obstetrics.
*Houston Everett, M.D., Instructor in Gynecology.
‡Edward G. Boettiger, Ph.D., Instructor in Physiology.
Murray M. Copeland, M.D., Instructor in Surgery.
J. Duer Moores, B.S., M.D., Instructor in Surgery.
†Philip S. Wagner, M.D., Instructor in Psychiatry.

*Irving Freeman, B.S., M.D., Instructor in Medicine.
†James C. Plagge, Ph.D., Instructor in Gross Anatomy.
†James U. Thompson, A.B., M.D., Instructor in Gross Anatomy.
†Walter H. Gerwig, Jr., M.D., Instructor in Physiology.

ASSISTANTS

William R. Geraghty, B.S., M.D., Assistant in Surgery.
Jaroslav Hulla, M.D., Assistant in Obstetrics.
Marius P. Johnson, A.B., M.D., Assistant in Pharmacology and Obstetrics.
Howard B. McElwain, M.D., Assistant in Surgery.
A. V. Buchness, A.B., M.D., Assistant in Surgery.
T. J. Touhey, M.D., Assistant in Surgery.
W. J. Schmitz, M.D., Assistant in Pediatrics.
John G. Runkle, M.D., Assistant in Ophthalmology.
Margaret B. Ballard, M.D., Assistant in Obstetrics.
Clyde F. Karns, B.S., M.D., Assistant in Surgery.
Morris Fine, M.D., Assistant in Medicine.
Samuel H. Culver, M.D., Assistant in Surgery.
W. H. Triplett, M.D., Assistant in Medicine.
E. Hollister Davis, A.B., M.D., Assistant in Anaesthesia.
Israel P. Meranski, B.S., M.D., Assistant in Pediatrics.
Albert R. Wilkerson, M.D., Assistant in Surgery.
Dudley P. Bowe, A.B., M.D., Assistant in Obstetrics.
Dorothy E. Schmalzer, B.S., Assistant in Biological Chemistry.
H. Whitney Wheaton, M.D., Assistant in Pediatrics.
Lauriston L. Keown, A.B., M.D., Assistant in Pediatrics.
J. H. Wilkerson, M.D., Assistant in Surgery.
L. T. Chance, M.D., Assistant in Surgery.
Samuel McLanahan, Jr., A.B., M.D., Assistant in Surgery.
Samuel E. Proctor, A.B., M.D., Assistant in Surgery.
George Govatos, A.B., M.D., Assistant in Surgery.
Jerome Snyder, B.S., M.D., Assistant in Ophthalmology.
William H. Grenzer, M.D., Assistant in Medicine.
George Silverton, A.B., M.D., Assistant in Medicine.
Dwight M. Currie, A.B., M.D., Assistant in Surgery.
Rollin C. Hudson, M.D., Assistant in Dermatology.
Thomas E. Roach, B.S., M.D., Assistant in Dermatology.
Milton C. Lang, M.D., Assistant in Ophthalmology.
Maxwell L. Mazer, M.D., Assistant in Obstetrics.
Joseph M. Blumberg, B.S., M.D., Assistant in Medicine and Obstetrics.
Hugh B. McNally, B.S., M.D., Assistant in Obstetrics.
Ferd. E. Kadan, A.B., M.D., Assistant in Obstetrics.
Lawrence Katzenstein, M.D., Assistant in Medicine.
Leon Ashman, M.D., Assistant in Medicine.
Nathaniel M. Beck, A.B., M.D., Assistant in Medicine and Gastro-Enterology.
John A. Myers, B.E.E., M.E.E., M.D., Assistant in Medicine and Gastro-Enterology.
Abraham Hurwitz, M.D., Assistant in Pediatrics.
Charles E. Balfour, M.D., Assistant in Neurology.
Robert F. Healy, M.D., Assistant in Surgery.
Herbert M. Foster, M.D., Assistant in Surgery.

Daniel R. Robinson, M.D., Assistant in Surgery.
H. L. Granoff, A.B., M.D., Assistant in Gynecology.
J. J. Erwin, M.D., Assistant in Gynecology.
Israel Rosen, A.B., M.D., Assistant in Pediatrics.
William Greenfeld, M.D., Assistant in Gastro-Enterology.
‡S. Edwin Muller, M.D., Assistant in Medicine.
†Edward F. Cotter, M.D., Assistant in Neurology.
Samuel Snyder, M.D., Assistant in Medicine.
Paul E. Carliner, B.S., M.D., Assistant in Medicine.
Kurt Levy, M.D., Assistant in Medicine.
William A. Parr, M.D., Assistant in Otology.
Cleo D. Stiles, M.D., Assistant in Diseases of the Nose and Throat and †Otology.
Jeannette R. Heghinian, B.S., M.D., Assistant in Dermatology.
Joseph V. Jerardi, B.S., M.D., Assistant in Surgery.
Thurston R. Adams, M.D., Assistant in Surgery.
Robert F. Chenowith, M.D., Assistant in Surgery.
Jacob R. Jensen, B.S., M.D., Assistant in Obstetrics.
J. Edward Norris, M.D., Assistant in Obstetrics.
J. Warren Albrittain, B.S., M.D., Assistant in Obstetrics.
‡Roland E. Bieren, M.D. Assistant in Pathology.
Karl F. Mech, B.S., M.D., Assistant in Gross Anatomy.
Henry L. Wollenweber, M.D., Assistant in Pathology.
Joye E. Jacobs, A.B., Assistant in Physiology.
†Howard B. Mays, M.D., Assistant in Genito-Urinary Surgery.
†Hugh Jewett, M.D., Assistant in Genito-Urinary Surgery.
†John V. Hopkins, M.D., Assistant in Orthopaedic Surgery.
†Eldred Roberts, B.S., M.D., Assistant in Surgery.
†Arthur G. Siwinski, A.B., M.D., Assistant in Surgery.
†Alexander McGill Duff, M.D., Assistant in Surgery.
†Lester N. Kolman, M.D., Assistant in Dermatology.
Louis J. Kroll, A.B., M.D., Assistant in Medicine.
William G. Helfrich, B.S., M.D., Assistant in Medicine.
†Frederick J. Vollmer, B.S., M.D., Assistant in Medicine.
†John L. Atkins, A.B., M.D., Assistant in Medicine.
†Gibson J. Wells, A.B., M.D., Assistant in Pediatrics.
†Harry B. Teitelbaum, B.S., M.D., Assistant in Neurology.
†John W. Machen, M.D., Assistant in Neurology.
†Arnold L. Peter, M.D., Assistant in Ophthalmology.

FELLOWS

Mildred Donohue, B.S., Weaver Fellow in Histology.
J. Victor Monke, M.S., Weaver Fellow in Physiology.
Frederick K. Bell, Ph.D., U. S. Pharmacopoeia Fellow.
Sylvan Forman, M.S., Emerson Fellow in Pharmacology.
Fred W. Ellis, M.S., Fellow in Pharmacology.
Frances F. Beck, Ph.D., International Cancer Research Foundation Fellow in Pharmacology.
†John Atkins, A.B., M.D., Weaver Fellow in Pathology.
‡Milton S. Sacks, B.S., M.D., Hitchcock Fellow in Medicine.
William K. Waller, A.B., Clinical Fellow in Medicine.

‡†Otto C. Brantigan, M.D., ‡Hitchcock Fellow in Surgery.
†Weaver Fellow in Gross Anatomy.
Edward G. Boettiger, Ph.D., Hitchcock Fellow in Gynecology.
Gordon M. Stephens, A.B., M.D., Fellow in Psychiatry.
‡Mabel G. Wilkin, M.A., M.D., Clinical Fellow in Pediatrics.
Alexander McGill Duff, M.D., Fellow National Cancer Institute.
Arthur G. Siwinski, A.B., M.D., Fellow National Cancer Institute (in absentia).
Reuben Rochkind, A.B., M.D., Fellow in Dermatology.
†Edward F. Cotter, M.D., Hitchcock Fellow in Neuro-Surgery.
†Ephraim T. Lisansky, A.B., M.D., Weaver Fellow in Pathology.
†Wilson C. Grant, M.S., International Cancer Research Foundation Research Assistant in Pharmacology.
†Nellie Cone, A.B., Emerson Fellow in Pharmacology.
†Edward Merdinyan, M.S., Emerson Fellow in Pharmacology.
†Howard B. Mays, M.D., Fellow in Urology and Biological Chemistry.

This list, and all subsequent lists, give the names of the members of the instruction staffs and their positions for 1939–1940 and 1940–1941, unless otherwise indicated.

Interim changes in names and positions are noted as follows:

* Promotions effective 1940–1941.
† Appointments effective 1940–1941.
‡ Resignations effective end of 1939–1940.
§ Deaths—dates of.

UNIVERSITY HOSPITAL COUNCIL

Harry Clifton Byrd, *President.*
W. W. Skinner, *Chairman of the Board of Regents.*
H. Boyd Wylie, *Acting Dean of the School of Medicine.*
M. C. Pincoffs, *Head of the Department of Medicine.*
A. M. Shipley, *Head of the Department of Surgery.*
J. Mason Hundley, Jr., *Head of the Department of Gynecology.*
Louis H. Douglass, *Head of the Department of Obstetrics.*
Clyde A. Clapp, *Head of the Department of Ophthalmology.*
[1]A. J. Lomas, *Superintendent of the Hospital.*
Miss Annie Crighton, R.N., *Superintendent of Nurses.*
Mrs. R. B. Pue, *Representing Woman's Auxiliary Board.*
Mrs. John L. Whitehurst, *Member Board of Regents.*

Harry M. Stein Charles A. Reifschneider	*Representing Hospital Staff.*
G. Carroll Lockard Alfred T. Gundry	*Representing Medical Alumni.*

UNIVERSITY HOSPITAL STAFF

[1]A. J. Lomas, *Superintendent*

Physician-in-Chief	Maurice C. Pincoffs
Physicians	Harry M. Stein Walter A. Baetjer Thomas R. Sprunt John E. Legge L. A. M. Krause G. Carroll Lockard Joseph E. Gichner William H. Smith William S. Love, Jr. T. Nelson Carey Raymond Hussey
Gastro-Enterologist	Zachariah R. Morgan
Neurologists	Irving J. Spear George M. Settle Leon Freedom
Psychiatrists	Ross McC. Chapman Ralph P. Truitt Harry M. Murdock
Pediatricians	C. Loring Joslin Albert Jaffe †J. E. Bradley
Pathologist	Hugh R. Spencer
Surgeon-in-Chief	Arthur M. Shipley

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[1] Retired December 31, 1939.

Surgeons	Charles Reid Edwards Page Edmunds Thomas B. Aycock
Neurological Surgeons	Charles Bagley, Jr. Richard G. Coblentz
Laryngologists	Edward A. Looper Franklin B. Anderson Thomas R. O'Rourk
Proctologists	J. Dawson Reeder Monte Edwards
Orthopaedic Surgeons	Allen Fiske Voshell Moses Gellman Compton Riely Henry F. Ullrich
Genito-Urinary Surgeons	W. H. Toulson W. A. H. Councill Lyle J. Millan Harry S. Shelley Howard B. Mays
Roentgenologists	Henry J. Walton Eugene L. Flippin Walter L. Kilby
Dermatologist	Harry M. Robinson
Bronchoscopist	Edward A. Looper
Otologist	J. W. Downey, Jr.
Anaesthetists	S. Griffith Davis E. Hollister Davis Mary J. O'Brien, R.N. Ruth Elliott, R.N.
Obstetricians	L. H. Douglass M. A. Novey Isadore A. Siegel J. G. M. Reese J. E. Savage
Ophthalmologists	Clyde A. Clapp William Tarun Henry F. Graff
Gynecologists	J. M. Hundley, Jr. R. G. Willse Leo Brady
Oncologists	J. M. Hundley, Jr. Grant E. Ward

ANNUAL HOSPITAL APPOINTMENTS

Each session the following annual appointments are made from among the graduates of the school to the University Hospital:

- Seven Residents in Surgery
- Four Residents in Medicine
- Four Residents in Obstetrics
- Two Residents in Gynecology
- Three Residents in Pediatrics
- Resident in Nose & Throat
- Three Residents in Roentgenology
- Twelve Senior Internes rotating in Medicine and Surgery
- Twelve Junior Internes rotating in the Specialties

UNIVERSITY HOSPITAL RESIDENT AND INTERN STAFF
JULY 1, 1939 TO JUNE 30, 1940

RESIDENT STAFF

Walter H. Gerwig, Jr., M.D., *Resident Surgeon.*
Harry C. Bowie, B.S., M.D., *First Assistant Resident Surgeon.*
Stuart G. Coughlan, B.S., M.D., *Assistant Resident Surgeon.*
D. Frank O. Kaltreider, Jr., A.B., M.D., *Assistant Resident Surgeon.*
William B. Long, Jr., B.S., M.D., *Assistant Resident Surgeon.*
James R. Martin, M.D., *Assistant Resident Surgeon.*
Edward A. Dunlap, M.D., *Assistant Resident Surgeon assigned to Eye, Ear, Nose and Throat.*
William W. McKinney, M.D., *Assistant Resident Surgeon assigned to Neuro-Surgery.*
James G. Arnold, Jr., A.B., M.D., *Surgical Intern.*
Stephen L. Magness, A.B., M.D., *Surgical Intern.*
Edward F. Cotter, M.D., *Resident Physician.*
Samuel T. R. Revell, Jr., B.S., M.D., *Assistant Resident Physician.*
Joseph E. Muse, M.D., *Assistant Resident Physician.*
Ephraim T. Lisansky, M.D., *Assistant Resident Physician.*
James Upshur Thompson, B.A., M.D., *Medical Intern.*
Darius M. Dixon, M.D., *Resident Obstetrician.*
John Dorman, M.D., *Assistant Resident Obstetrician.*
M. M. Schneider, M.D., *Assistant Resident Obstetrician.*
John K. B. E. Seegar, Jr., A.B., M.D., *Assistant Resident Obstetrician.*
William B. Settle, M.D., *Resident Gynecologist.*
Everett S. Diggs, B.S., M.D., *Assistant Resident Gynecologist.*
Gibson J. Wells, A.B., M.D., *Resident Pediatrician.*
Robert J. Gore, M.D., *Assistant Resident Pediatrician.*
Geraldine Powell, M.D., *Assistant Resident Pediatrician.*
Alston E. Morrison, M.D., *Resident Roentgenologist.*
Charles N. Davidson, M.D., *Assistant Resident Roentgenologist.*

SENIOR INTERNS

Stanley E. Bradley, A.B., M.D.
John Z. Bowers, B.S., M.D.
John J. Bunting, M.D.
Timothy A. Callahan, Jr., M.D.
Louis C. Gareis, M.D.
Joseph M. George, Jr., A.B., M.D.
John H. Haase, M.D.
Samuel Novey, M.D.
Conover Talbot, A.B., M.D.
Bernard O. Thomas, B.S., M.D.
John A. Wagner, B.S., M.D.
Theodore E. Woodward, B.S., M.D.

JUNIOR INTERNS

Henry A. Briele, M.D.
Raymond M. Cunningham, A.B., M.D.
Leo J. Gaver, M.D.
Alvin S. Hartz, A.B., M.D.
William H. Leitch, B.S., M.D.
Francis J. McLaughlin, M.D.
William S. Miller, A.B., M.D.
James B. Nuttall, M.D.
Leslie H. Pierce, B.S., M.D.
Edwin R. Ruzicka, B.S., M.D.
Leland B. Stevens, B.S., M.D.
Ramsay B. Thomas, B.S., M.D.

DENTAL INTERN

Dorsey R. Tipton, D.D.S.

ANNOUNCEMENT OF UNIVERSITY HOSPITAL RESIDENT AND INTERN STAFF EFFECTIVE JULY 1, 1940

RESIDENT STAFF

Harry C. Bowie, B.S., M.D., *Resident Surgeon.*
Stuart G. Coughlan, B.S., M.D., *First Assistant Resident Surgeon.*
William B. Long, Jr., B.S., M.D., *First Assistant Resident Surgeon.*
John H. Haase, M.D., *Assistant Resident Surgeon.*
Robert C. Sheppard, M.D., *Assistant Resident Surgeon.*
Robert L. Gottschalk, A.B., M.D., *Assistant Resident Surgeon.*
Bernard Rabold, B.S., M.D., *Assistant Resident Surgeon.*
James G. Arnold, Jr., A.B., M.D., *Assistant Resident Surgeon assigned to Neuro-Surgery.*
William C. Humphries, A.B., M.D., *Assistant Resident Surgeon assigned to Eye, Ear, Nose and Throat.*
Stephen L. Magness, A.B., M.D., *Surgical Intern.*
John L. Atkins, A.B., M.D., *Resident Physician.*
John Z. Bowers, B.S., M.D., *Assistant Resident Physician.*
Timothy A. Callahan, Jr., M.D., *Assistant Resident Physician.*
Conover Talbot, A.B., M.D., *Assistant Resident Physician.*
John K. B. Seegar, Jr., A.B., M.D., *Resident Obstetrician*
M. M. Schneider, M.D., *Assistant Resident Obstetrician.*
Louis C. Gareis, M.D., *Assistant Resident Obstetrician.*
Robert Linn, M.D., *Assistant Resident Obstetrician.*
Everett S. Diggs, B.S., M.D., *Resident Gynecologist.*
William K. Diehl, B.S., M.D., *Assistant Resident Gynecologist.*
Roland E. Bieren, M.D., *Assistant Resident Gynecologist.*
Charles N. Davidson, M.D., *Resident Roentgenologist.*
George W. McCall, A.B., M.D., *Assistant Resident Roentgenologist.*
Robert J. Gore, A.B., M.D., *Resident Pediatrician.*
Earl S. Scott, M.D., *Assistant Resident Pediatrician.*
Elizabeth Bess, A.B., M.D., *Assistant Resident Pediatrician.*

SENIOR INTERNS

Henry A. Briele, M.D.
Raymond M. Cunningham, A.B., M.D.
Leo J. Gaver, M.D.
Alvin S. Hartz, A.B., M.D.
William H. Leitch, B.S., M.D.
Francis J. McLaughlin, M.D.
William S. Miller, A.B., M.D.
James B. Nuttall, M.D.
Leslie H. Pierce, B.S., M.D.
Edwin R. Ruzicka, B.S., M.D.
Leland B. Stevens, B.S., M.D.
Ramsay B. Thomas, B.S., M.D.

JUNIOR INTERNS

Lester H. Caplan, A.B., M.D.
Edwin O. Daue, B.S., M.D.
R. L. Gibbs, Jr., A.B., M.D.
Walter R. Graham, A.B., M.D.
Benjamin H. Inloes, Jr., M.D.
James R. Karns, M.D.
H. Pearce Maccubbin, B.S., M.D.
Robert B. Mearns, M.D.
C. Martin Rhode, A.B., M.D.
Alexander F. Thompson, A.B., M.D.
Wilfred H. Townshend, Jr., A.B., M.D.
Richard T. Williams, A.B., M.D.

UNIVERSITY HOSPITAL DISPENSARY STAFF

Emma Winship, R.N.
Dispensary Director

DISPENSARY COMMITTEE

John E. Legge, *Chairman*
Emma Winship, R.N., *Secretary*

L. H. Douglass
J. Mason Hundley, Jr.
C. Loring Joslin
John E. Savage
George H. Yeager
[1]A. J. Lomas
Ralph P. Truitt

Chief of Medical Clinic	John E. Legge
Assistant Chiefs of Medical Clinic	Lewis P. Gundry Irving Freeman
Physicians	Harry G. Jones Charles H. Reier M. A. Fine Herbert Goldstone Nachman Davidson George Silverton Carl P. Roetling Albert J. Himelfarb Kurt Levy ‡George J. Sawyer ‡George H. Davis
Chief of Gastro-Enterology Clinic	Zachariah Morgan
Assistant Gastro-Enterologists	Z. V. Hooper Ernest Levi John A. Myers M. S. Koppelman Samuel Morrison Albert J. Shochat William Greenfeld

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[1] Retired December 31, 1939.

Chief of Neurology Clinic	Leon Freedom
Assistant Neurologists	Benjamin Pushkin Bernhard Badt William L. Fearing Charles E. Balfour †H. A. Teitelbaum
Chief of Psychiatric Clinic	Ralph P. Truitt
Assistant Psychiatrists	H. W. Newell Harry Goldsmith Harry Murdock Alice J. Rockwell Gordon M. Stephens
Chief of Chest Clinic	E. H. Tonolla
Assistants, Diseases of the Lungs	H. V. Langeluttig Manuel Levin Meyer W. Jacobson †John Constantini
Chief of Metabolism Clinic	Lewis P. Gundry
Assistants	William G. Helfrich Walter E. Karfgin A. Goldman
Chief of Cardiovascular Clinic	William S. Love, Jr.
Assistant Chiefs of Cardiovascular Clinic	Robert W. Garis *Robert B. Mitchell
Assistant Cardiologists	Samuel J. Hankin Anthony F. Carozza W. G. Hersperger Richard S. Owens Irving Freeman Harry M. Robinson, Jr.
Chief of Allergy Clinic	H. M. Bubert
Assistant Chiefs of Allergy Clinic	Edward S. Kallins †Richard S. Owens
Assistant Allergists	Paul E. Carliner Nachman Davidson Samuel Snyder Benjamin S. Rich Frank V. Manieri Irving Freeman
Allergy Clinic Technician	Selma Rosenberg
Director, Pediatric Clinic	C. Loring Joslin
Chief of Pediatric Clinic	John H. Traband
Chief of Pediatric Clinic	Clewell Howell

Assistant Pediatricians	M. Paul Byerly A. H. Finkelstein Samuel S. Glick F. Strattner Orem Israel P. Meranski Robert B. Taylor William M. Seabold Jerome Fineman H. Whitney Wheaton Albert Jaffe S. Lilienfeld Thomas A. Christensen Lauriston L. Keown Israel Rosen Abraham B. Hurwitz Joseph R. Meyerowitz Louis R. Maser Gibson J. Wells Robert J. Gore †Elizabeth Bess
Chief of Surgical Clinic	George H. Yeager
Assistant Surgeons	J. Frank Hewitt Thurston R. Adams Luther E. Little George H. Brouillet W. R. Johnson Karl F. Mech Samuel E. Proctor C. Vernon Williamson A. R. Wilkerson Robert Chenowith
Chief of Orthopaedic Surgery Clinic	Allen Fiske Voshell
Assistant Orthopaedic Surgeons	John V. Hopkins Moses Gellman Henry F. Ullrich Fred Duckwall
Chief of Genito-Urinary Clinic	W. H. Toulson
Assistant Genito-Urinary Surgeons	W. A. H. Councill John F. Hogan L. J. Millan Harry S. Shelley Howard B. Mays
Chief of Roentgenology Clinic	Henry J. Walton
Assistant Roentgenologist	Eugene L. Flippin
Chief of Dermatology Clinic	Harry M. Robinson

Assistant Dermatologists	Rollin C. Hudson Joseph C. Bernstein Thomas E. Roach Harold M. Goodman Harry M. Robinson, Jr. Jeannette R. Heghinian Lester N. Kolman Reuben Rochkind
Chief of Rhinology and Laryngology Clinic	Franklin B. Anderson
Assistant Rhinologists and Laryngologists	Joseph Nurkin Cleo D. Stiles Thomas R. O'Rourk Benjamin S. Rich M. Baylus
Chief of Proctology Clinic	Monte Edwards
Assistant Proctologist	James C. Owings
Chief of Gynecology Clinic	J. Mason Hundley, Jr.
Assistant Chief of Gynecology Clinic	Beverley C. Compton
Assistant Gynecologists	Thomas S. Bowyer Joseph V. Castagna John C. Dumler John T. Hibbitts C. Bernard Brack Kenneth B. Boyd W. Allen Deckert Marius P. Johnson Hugh B. McNally Charles B. Marek Helen I. Maginnis
Female Cystoscopists	J. Mason Hundley, Jr. W. Allen Deckert Beverley C. Compton John C. Dumler
Chief of Oral Surgery Clinic	Brice M. Dorsey
Oral Surgeons	Harold Goldstein Samuel H. Bryant
Chief of Obstetrical Clinic	John E. Savage
Assistant Obstetricians	M. B. Ballard Maxwell L. Mazer Jaroslav Hulla Marius P. Johnson Dudley P. Bowe W. Allen Deckert Joseph M. Blumberg Hugh B. McNally Ferd E. Kadan Jacob R. Jensen J. Edward Norris J. Warren Albrittain

Oncology Clinic, Gynecological Division	J. Mason Hundley, Jr. Thomas S. Bowyer Beverley C. Compton John C. Dumler
Oncology Clinic, Surgical Division	Grant E. Ward Eugene E. Covington J. Duer Moores J. W. Nelson Eldred Roberts Murray M. Copeland Alexander McHill Duff Arthur G. Siwinski
Chief of Ophthalmology Clinic	H. F. Graff
Assistant Ophthalmologists	Frank A. Holden Thomas R. O'Rourk Milton C. Lang John G. Runkle Jerome Snyder †Arnold L. Peter
Directress, Occupational Therapy	Miss Nancy Valliant
Directress, Social Service	Miss Grace Pearson

UNIVERSITY HOSPITAL DISPENSARY REPORT

October 1, 1938 to September 30, 1939

Departments	*New Cases*	*Old Cases*	*Total*
Allergy Clinic	216	5,402	5,618
Cardiology	337	2,110	2,447
Curative Workshop	38	1,149	1,187
Cystoscopy	118	473	591
Dermatology	4,583	16,215	20,798
Diabetic	84	1,269	1,353
Gastro-Intestinal	179	1,252	1,431
Genito-Urinary	643	3,185	3,828
Gynecology	1,910	5,279	7,189
Medicine	2,379	5,095	7,474
Neurology	265	1,114	1,379
Nose, Throat and Ear	1,638	1,335	2,973
Obstetrics	1,599	10,055	11,654
Oncology	363	2,338	2,701
Ophthalmology	1,485	3,872	5,357
Oral Surgery	1,053	834	1,887
Orthopaedic	1,699	4,227	5,926
Pediatrics	2,536	12,829	15,365
Physiotherapy	261	3,140	3,401
Proctology	270	452	722
Psychiatry	86	312	398
Surgery	2,743	8,383	11,126
Tuberculosis	241	2,372	2,613
Total	24,726	92,692	117,418

MERCY HOSPITAL

BOARD OF GOVERNORS

[1]Walter D. Wise, *Chairman*

Sister M. Placide
Sister M. Veronica
Sister M. Hildegarde
Sister M. de Chantal
Sister M. Hilda

[1]Henry F. Bongardt
Maurice C. Pincoffs
Waitman F. Zinn
Thomas K. Galvin
Edward P. Smith

Sister M. Joseph

MERCY HOSPITAL STAFF

Consulting Surgeon	§Alexius McGlannan
Surgeon-in-Chief	Walter D. Wise
Surgeons	Charles F. Blake F. L. Jennings R. W. Locher Thomas R. Chambers Elliott H. Hutchins D. J. Pessagno William F. Rienhoff N. Clyde Marvel H. F. Bongardt
Associate Surgeons	I. O. Ridgely J. W. Nelson Howard B. McElwain Simon H. Brager John A. O'Connor Thomas B. Aycock Charles W. Maxson I. Ridgeway Trimble R. T. Shackelford Raymond F. Helfrich
Assistant Surgeons	Julius Goodman Eugene E. Covington S. Demarco, Jr. T. J. Touhey William N. McFaul, Jr. Meyer H. Zuravin Howard L. Zupnik Daniel R. Robinson Joseph V. Jerardi
Consulting Ophthalmologist and Otologist	Harry Friedenwald
Ophthalmologists and Otologists	H. K. Fleck J. W. Downey M. Raskin

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[1] Appointed April 9, 1940.

§ Deceased, February 25, 1940.

Associate Ophthalmologists and Otologists	Joseph I. Kemler F. A. Pacienza
Assistant Ophthalmologist and Otologist	Joseph V. Jeppi
Consulting Rhinologists and Laryngologists	W. Raymond McKenzie George W. Mitchell
Rhinologist and Laryngologist	Waitman F. Zinn
Associate Rhinologists and Laryngologists	F. A. Pacienza F. A. Kayser Benjamin S. Rich Theodore A. Schwartz Birkhead Macgowan
Assistant Rhinologist and Laryngologist	Joseph V. Jeppi
Bronchoscopist	Waitman F. Zinn
Associate Bronchoscopist	F. A. Kayser
Assistant Bronchoscopist	Theodore A. Schwartz
Proctologist	Charles F. Blake
Assistant Proctologist	Eugene E. Covington
Orthopaedic Surgeon	Albertus Cotton
Associate Orthopaedic Surgeon	H. L. Rogers
Assistant Orthopaedic Surgeons	I. H. Maseritz J. H. Gaskel
Urologist	Kenneth D. Legge
Associate Urologists	Leon K. Fargo Francis W. Gillis
Dentist	J. D. Fusco
Assistant Dentists	J. J. Foley W. Inman
Physician-in-Chief	Maurice C. Pincoffs
Physicians	C. B. Gamble Harvey G. Beck Thomas P. Sprunt H. Raymond Peters George McLean J. Sheldon Eastland L. A. M. Krause
Associate Physicians	Hubert C. Knapp Bartus T. Baggott Thomas C. Wolff Wetherbee Fort John E. Legge T. Nelson Carey
Assistant Physicians	Sol Smith S. A. Tumminello J. Howard Burns Earl L. Chambers K. W. Golley J. M. Miller

Gastro-Enterologist	Julius Friedenwald
Associate Gastro-Enterologists	T. Frederick Leitz Theodore H. Morrison
Assistant Gastro-Enterologists	Maurice Feldman Joseph Sindler
Pediatricians	Edgar B. Friedenwald *Frederick B. Smith
Associate Pediatricians	Thomas J. Coonan *G. Bowers Mansdorfer
Assistant Pediatricians	William J. Schmitz William M. Seabold
Neurologist and Psychiatrist	Andrew C. Gillis
Associate Neurologists and Psychiatrists	Harry Goldsmith Philip F. Lerner §Milford Levy
Obstetricians	Edward P. Smith Abram S. Samuels Ernest S. Edlow William S. Gardner Thomas K. Galvin John J. Erwin
Associate Obstetrician	Frank K. Morris
Assistant Obstetrician	William B. Smith
Consulting Gynecologist	William S. Gardner
Gynecologists	Abram S. Samuels Thomas K. Galvin Edward P. Smith George S. Strauss, Jr.
Associate Gynecologists	J. J. Erwin Ernest S. Edlow Frank K. Morris
Assistant Gynecologist	Harry L. Granoff
Pathologists	Walter C. Merkel Hugh R. Spencer
Clinical Pathologists	H. T. Collenberg H. R. Peters Charles E. Bramble
Technicians	Sister Mary Celeste Eleanor Behr Anna Wassell Audrey Auten
Radiographer	Albertus Cotton
Associate Radiographer	Harry L. Rogers
Assistant Radiographer	Harry Archer Miller
Technicians	Sister M. Kevin Elizabeth Crook

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§ Deceased, October 10, 1939.

ANNUAL HOSPITAL APPOINTMENTS

Each session the following annual appointments are made from among the graduates of the school to the Mercy Hospital:

Five Residents in Surgery
Resident in Rhinology
Two Residents in Medicine
Two Residents in Gynecology
Ten Interns on Rotating Service

MERCY HOSPITAL RESIDENT AND INTERN STAFF JULY 1, 1939 TO JUNE 30, 1940

Raymond J. Lipin, M.D., *Resident Surgeon.*
W. Lynn Garlick, A.B., M.D., *First Assistant Resident Surgeon.*
Robert W. Stevens, B.S., M.D., *Resident Gynecologist.*
William A. Dodd, B.S., M.D., *Assistant Resident Gynecologist.*
C. H. Doeller, A.B., M.D., *Resident Obstetrician.*
William C. Dunnigan, A.B., M.D., *Second Assistant Resident Surgeon.*
John F. Schaefer, B.S., M.D., *Second Assistant Resident Surgeon.*
William B. Culwell, B.S., M.D., *Second Assistant Resident Surgeon.*
S. Edwin Muller, M.D., *Resident Physician.*
Donald D. Cooper, A.B., M.D., *Assistant Resident Physician.*
Frederick J. Vollmer, B.S., M.D., *Resident in Pathology and Assistant Resident Physician.*
Maynard C. Smith, B.S., M.D., *Resident Rhinologist.*

INTERNS

Harry McBrine Beck, A.B., M.D.
Richard W. Corbitt, A.B., M.D.
George Smith Grier, B.S., M.D.
William H. Kammer, Jr., A.B., M.D.
Howard F. Kinnamon, Jr., M.D.
Christopher J. Mendelis, B.S., M.D.
Philip J. Morrison, B.S., M.D.
Melvin F. Polek, B.S., M.D.
Thomas S. Sexton, A.B., M.D.
Joseph E. Schenthal, A.B., M.D.

ANNOUNCEMENT OF MERCY HOSPITAL RESIDENT AND INTERN STAFF

EFFECTIVE JULY 1, 1940

W. Lynn Garlick, A.B., M.D., *Resident Surgeon.*
John F. Schaefer, B.S., M.D., *Associate Resident Surgeon.*
Howard F. Kinnamon, Jr., M.D., *Assistant Resident Surgeon.*
Richard W. Corbitt, A.B., M.D., *Assistant Resident Surgeon.*
Melvin F. Polek, B.S., M.D., *Assistant Resident Surgeon.*
Frederick J. Vollmer, B.S., M.D., *Resident Physician.*
William H. Kammer, Jr., A.B., M.D., *Assistant Resident Physician.*
John A. Wagner, B.S., M.D., *Assistant Resident Physician.*
Joseph E. Schenthal, A.B., M.D., *Assistant Resident Physician and Resident Pathologist.*
Charles H. Doeller, Jr., A.B., M.D., *Resident Gynecologist.*
William A. Dodd, B.S., M.D., *Resident Obstetrician.*
Harry McBrine Beck, A.B., M.D., *Assistant Resident Gynecologist and Obstetrician.*
Maynard C. Smith, B.S., M.D., *Resident Rhinologist.*

INTERNS

George B. Barnes, M.D.
Paul H. Correll, A.B., M.D.
William C. Duffy, A.B., M.D.
James A. Freeman, M.D.
William C. Livingood, B.S., M.D.
F. Ford Loker, B.S., M.D.
William D. McClung, M.D.
Forest C. Meade, M.D.
Donald J. Roop, A.B., M.D.
William E. Weeks, B.S., M.D.

MERCY HOSPITAL DISPENSARY STAFF

Supervisor of Surgical Clinic	Raymond F. Helfrich
Assistant Surgeons	I. O. Ridgely H. F. Bongardt J. W. Nelson I. Ridgeway Trimble Simon H. Brager S. Demarco, Jr. Eugene E. Covington Richard T. Shackelford Meyer H. Zuravin Howard L. Zupnik Daniel R. Robinson Joseph V. Jerardi
Supervisor of Genito-Urinary Clinic	Kenneth D. Legge
Assistant Genito-Urinary Surgeons	L. K. Fargo Francis W. Gillis
Orthopaedic Surgeons	Albertus Cotton Harry L. Rogers I. H. Maseritz Jason H. Gaskel
Supervisor of Medical Clinic	Maurice C. Pincoffs
Chiefs of Medical Clinic	Earl L. Chambers Sol Smith
Assistant Physicians	Milton Stein ‡William H. Grenzer Thomas G. Abbott Louis F. Klimes ‡John W. Machen ‡William G. Helfrich †Irving Freeman
Chief of Cardiovascular Clinic	Thomas C. Wolff
Assistant Cardiologist	Harry Ashman
Chief of Metabolism Clinic	J. Sheldon Eastland
Assistant	Louis F. Klimes
Supervisor of Gastroenterology Clinic	Julius Friedenwald
Gastroenterologists	Maurice Feldman Nathaniel Beck
Chief of Pediatric Clinic	Edgar Friedenwald

Pediatricians	G. Bowers Mansdorfer Jerome Fineman George Bayley
Supervisor of Neurological and Psychiatric Clinic	Andrew C. Gillis
Neurologists and Psychiatrists	§Milford Levy Harry Goldsmith Philip F. Lerner †John W. Machen
Director of Child Guidance Clinic	Miriam Dunn
Supervisor of Dermatology Clinic	Francis A. Ellis
Dermatologist	William D. Wolfe
Oncologist	James W. Nelson
Chief of Gynecology Clinic	Abram S. Samuels
Assistant Chief of Gynecology Clinic	Thomas K. Galvin
Gynecologists	Edward P. Smith Ernest S. Edlow H. L. Granoff John J. Erwin Frank K. Morris George A. Strauss, Jr.
Chief of Obstetrical Clinic	Edward P. Smith
Obstetricians	William B. Smith Robert W. Stevens
Esophagoscopist	Waitman F. Zinn
Rhinologists and Laryngologists	Waitman F. Zinn Benjamin S. Rich Theodore A. Schwartz
Ophthalmologists and Otologists	M. Raskin F. A. Pacienza J. V. Jeppi
Supervisor of Proctology Clinic	Charles F. Blake
Proctologists	Eugene E. Covington Simon H. Brager
Dentists	J. D. Fusco W. Inman
Supervisor of Physiotherapy Clinic	Leon Hannan
Assistant Physiotherapist	Alice R. Hannan
Social Worker	Elmer Lambden
Secretaries	Norman Foxman Eleanor Arnick

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§ Deceased, October 10, 1939.

MERCY HOSPITAL DISPENSARY REPORT

Year of 1939

Department	*New Cases*	*Old Cases*	*Total*
Cardiac	27	307	334
Dental	144	135	279
Diabetic	24	291	315
Eye and Ear	342	738	1,080
Gastro-Intestinal	85	465	550
Genito-Urinary	272	1,939	2,211
Gynecology	300	912	1,212
Medicine	674	2,589	3,263
Neurology	89	613	702
Nose and Throat	609	1,317	1,926
Orthopaedics	214	898	1,112
Pediatrics	205	407	612
Physiotherapy	96	1,157	1,253
Postnatal	130	3	133
Prenatal	237	1,167	1,404
Psychiatry	18	68	86
Rectal	56	96	152
Skin	277	529	806
Surgery	1,229	4,725	5,954
Tumor	11	27	38
Well Clinic	194	108	302
	5,233	18,491	23,724

THE BALTIMORE CITY HOSPITALS

STAFF OF BALTIMORE CITY HOSPITALS

Parker J. McMillin, *Superintendent*

Consulting Chief Surgeon	Arthur M. Shipley, M.D., Sc.D.
Surgeon-in-Chief	Thomas B. Aycock, B.S., M.D.
Physician-in-Chief	John T. King, Jr., A.B., M.D.
Physician-in-Chief, Tuberculosis Hospital	Harry M. Stein, M.D.
Physician-in-Chief, Psychopathic Hospital	Esther L. Richards, M.D.
Obstetrician-in-Chief	Louis H. Douglass, M.D.
Pediatrician-in-Chief	T. Campbell Goodwin, A.B., M.D.
Pathologist-in-Chief	Frank B. Kindell, A.B., M.D.
Dental Surgeon-in-Chief	Lowell P. Henneberger, D.D.S.
Assistant Surgeon-in-Chief	Otto C. Brantigan, M.D.
Visiting Surgeon	James C. Owings, M.D.
Consultant in Traumatic Surgery	Charles A. Reifschneider, M.D.
Consultant in General Surgery	I. Ridgeway Trimble, M.D.
Consultant in Peripheral Vascular Diseases	George H. Yeager, B.S., M.D.
Consultant in Plastic Surgery	Edward M. Hanrahan, Jr., M.D.
Assistant Visiting Surgeons	Samuel McLanahan, Jr., A.B., M.D. Harry C. Hull, M.D. Luther E. Little, M.D.
Visiting Physician in Tuberculosis	Lawrence M. Serra, M.D.

Assistant Visiting Physician in Tuberculosis	HARRY V. LANGELUTTIG, A.B., M.D.
Assistant Visiting Psychiatrist	THOMAS A. C. RENNIE, M.D.
Visiting Obstetrician	J. G. MORRIS REESE, M.D.
Assistant Visiting Obstetricians	JOHN M. HAWS, B.S., M.D. MARIUS P. JOHNSON, A.B., M.D. JOHN E. SAVAGE, B.S., M.D. H. B. MCNALLY, B.S., M.D. JOHN WHITRIDGE, JR., A.B., M.D.
Visiting Pediatrician	B. MATTHEW DEBUSKEY, M.D.
Assistant Visiting Pediatrician	WILLIAM M. SEABOLD, A.B., M.D.
Visiting Gynecologist	J. MASON HUNDLEY, JR., M.A., M.D.
Assistant Visiting Gynecologists	JOHN T. HIBBITTS, M.D. JOHN C. DUMLER, B.S., M.D. BEVERLY COMPTON, M.D. BERNARD C. BRACK, M.D. ERNEST I. CORNBROOKS, M.D.
Assistant Chief Physician	C. HOLMES BOYD, M.D.
Visiting Physicians	RICHARD FRANCE, M.D. JOHN W. PARSONS, M.D. LOUIS A. M. KRAUSE, M.D.
Visiting Cardiologist	RAYMOND HUSSEY, M.A., M.D.
Visiting Ophthalmologist	FRANK B. WALSH, M.D.
Visiting Orthopaedic Surgeon	ALLEN F. VOSHELL, A.B., M.D.
Assistant Visiting Orthopaedic Surgeon	HENRY F. ULLRICH, M.D., Sc.D.
Assistant Visiting Laryngologists	THOMAS R. O'ROURK, M.D. JOHN BORDLEY, M.D.
Visiting Urologist	W. HOUSTON TOULSON, A.B., M.Sc., M.D.
Assistant Visiting Urologists	HUGH JEWETT, M.D. HARRY S. SHELLEY, B.S., M.D. HOWARD B. MAYS, M.D.
Chief Radiologist	JOHN W. PIERSON, M.D.
Assistant Visiting Radiologist	GEORGE J. FARBER, M.D.
Visiting Neurological Surgeon	CHARLES BAGLEY, JR., M.A., M.D.
Assistant Visiting Neurological Surgeon	RICHARD G. COBLENTZ, M.A., M.D.
Visiting Neurologists	FRANK R. FORD, M.D. O. R. LANGWORTHY, M.D.
Assistant Visiting Neurologists	LAWRENCE C. KOLB, M.D. FREDERICK H. HESSER, M.D. HARRY A. TEITELBAUM, M.D.
Visiting Proctologist	MONTE EDWARDS, M.D.
Visiting Oncologist	GRANT E. WARD, A.B., M.D.
Assistant Visiting Oncologist	MURRAY M. COPELAND, M.D.
Assistant Visiting Otologist	J. J. BELEMER, M.D.
Visiting Dermatologist	ISAAC R. PELS, M.D.
Visiting Dental Surgeon	H. GLENN WARING, D.D.S.

Assistant Visiting Dental Surgeons	Lawrence W. Bimestefer, D.D.S. Joseph B. Berke, D.D.S. B. W. Miksinski, D.D.S. Henry H. Hall, D.D.S.

THE JAMES LAWRENCE KERNAN HOSPITAL AND INDUSTRIAL SCHOOL OF MARYLAND FOR CRIPPLED CHILDREN

STAFF

Surgeon-in-Chief and Medical Director	Allen Fiske Voshell, A.B., M.D.
Attending Orthopaedic Surgeon and Roentgenologist	Albertus Cotton, A.M., M.D.
Associate Orthopaedic Surgeons	Moses Gellman, B.S., M.D. Harry Rogers, M.D. Winthrop M. Phelps, A.B., M.D. Henry F. Ullrich, M.D., Sc.D.
Plastic Surgeon	Edward A. Kitlowski, A.B., M.D.
Aurist and Laryngologist	Benjamin S. Rich, A.B., M.D.
Dentist	M. E. Coberth, D.D.S.
Pediatrist	J. Edmund Bradley, B.S., M.D.
Consulting Surgeons	J. M. T. Finney, A.B., M.D., D.S.M., F.R.C.S. (Eng., Ire.) Hon. Arthur M. Shipley, M.D., Sc.D. C. Reid Edwards, A.B., M.D. Thomas B. Aycock, B.S., M.D.
Consulting Aurist and Laryngologist	Franklin B. Anderson, M.D. Edward A. Looper, M.D., D.Oph.
Consulting Neurological Surgeon	Charles Bagley, Jr., M.A., M.D.
Consulting Plastic Surgeon	John Staige Davis, B.Sc., M.D.
Consulting Physicians	Thomas R. Brown, A.B., M.D. Lewellys F. Barker, A.B., M.D.
Consulting Dermatologists	Harry M. Robinson, M.D. Leon Ginsberg, M.D.
Consulting Neurologists	Irving J. Spear, M.D. R. V. Seliger, M.D.
Consulting Pediatrist	Benjamin Tappan, A.B., M.D.
Consulting Dentist	Harry B. McCarthy, D.D.S.
Consulting Opthalmologists	Harry Friedenwald, A.B., M.D., D.H.L., D.Sc. William Tarun, M.D.
Consulting Pathologist	Hugh R. Spencer, M.D.
Consulting Roentgenologists	Howard E. Ashbury, A.B., M.D. Henry Walton, M.D.
Resident Orthopaedic Surgeon	H. K. Magill, M.D.
Superintendent	Miss Maud M. Gardner, R.N.
Dispensary and Social Service Nurse	Miss Mabel S. Brown, R.N.
Anaesthetist	Miss Marie Bareford, R.N.

Physical Therapists and X-ray Technician	Miss Jane Ewing, P.T., Reg. Miss Jane Anderson, R.N. Mrs. Georgiana Wisong
Occupational Therapist	Miss Muriel Zimmerman, O.T.
Instructor in Grammar School	Mrs. Laura Siemon

HISTORY OF THE SCHOOL OF MEDICINE

The present School of Medicine, with the title University of Maryland School of Medicine and College of Physicians and Surgeons, is the result of a consolidation and merger of the University of Maryland School of Medicine with the Baltimore Medical College (1913) and the College of Physicians and Surgeons of Baltimore (1915).

The School of Medicine of the University of Maryland is one of the oldest foundations for medical education in America, ranking fifth in point of age among the medical colleges of the United States. It was organized in 1807, and chartered in 1808, under the name of the College of Medicine of Maryland, and its first class was graduated in 1810. In 1812 the College was empowered by the Legislature to annex three other colleges or faculties: Divinity, Law, and Arts and Sciences; and the four colleges thus united were "constituted an University by the name and under the title of the University of Maryland."

The original building of the Medical School at the N. E. corner of Lombard and Greene Streets, erected in 1812, is the oldest structure in America devoted to medical teaching. Here was founded one of the first medical libraries and the first medical college library in the United States.

At this Medical School for the first time in America, dissection was made a compulsory part of the curriculum, and independent chairs for the teaching of gynecology and pediatrics (1867), and of ophthalmology and otology (1873), were installed.

This School of Medicine was one of the first to provide for adequate clinical instruction by the erection of its own hospital in 1823. In this hospital intramural residency for senior students was established for the first time.

In 1913, a merger was brought about with the Baltimore Medical College, an institution of thirty-two years' growth. By this association the facilities of the School of Medicine were enlarged in faculty, equipment and hospital connection.

The College of Physicians and Surgeons was incorporated in 1872, and established on Hanover Street in a building afterward known as the *Maternité,* the first obstetrical hospital in Maryland. In 1878 union was effected with the Washington University School of Medicine, in existence since

1827, and the college was removed to Calvert and Saratoga Streets. By the consolidation with the College of Physicians and Surgeons, medical control of the teaching beds in the City Hospital, now the Mercy Hospital, was obtained.

BUILDINGS AND FACILITIES

The original medical building at the N. E. corner of Lombard and Greene Streets houses the office of the Dean, the Conference Room of the Faculty, and two lecture halls.

The Administration Building, to the east of the original building, contains the Baltimore offices of the Registrar and the Director of Admissions and two lecture halls.

The laboratory building at 31 South Greene Street is occupied by the departments of Pathology, Bacteriology and Biochemistry.

The Frank C. Bressler Research Laboratory provides the departments of Anatomy, Histology and Embryology, Pharmacology, Physiology and Clinical Pathology with facilities for teaching and research. It also houses the research laboratories of the clinical departments, animal quarters, a laboratory for teaching Operative Surgery, a lecture hall and the Bressler Memorial Room.

This building has been recently erected at 29 South Greene Street opposite the University Hospital. It was built with funds left to the School of Medicine by the late Frank C. Bressler, an alumnus, supplemented by a grant from the Federal government. The building, in the shape of an I, extends east from Greene Street, just north of the original building.

MEDICAL LIBRARIES

The Medical Library of the University of Maryland, founded in 1813 by the purchase of the collection of Dr. John Crawford, now numbers 20,000 volumes and several thousand pamphlets and reprints. Over three hundred of the leading medical journals, both foreign and domestic, are received regularly. The library is housed in Davidge Hall, a comfortable and commodious building in close proximity to classrooms and laboratories, and is open daily for the use of members of the faculty, the student body and the profession generally. Libraries pertaining to particular phases of medicine are maintained by several departments in the Medical School.

The library of the Medical and Chirurgical Faculty of Maryland and the Welch Medical Library are open to students of the Medical School without charge. Other libraries of Baltimore are the Peabody Library and the Enoch Pratt Free Library.

DISPENSARY BUILDING

The old hospital building has been remodeled and is occupied by the Out-patient Department. Thus the students have been provided with a splendidly appointed group of clinics for their training in out-patient work. All departments of clinical training are represented in this remodeled building and all changes have been predicated on the teaching function for which this department is intended.

The office of the Medical School Physician is located in this building.

The Department of Art also occupies quarters here.

UNIVERSITY HOSPITAL

The University Hospital, which is the property of the University of Maryland, is the oldest institution for the care of the sick in the State of Maryland. It was opened in September, 1823, under the name of the Baltimore Infirmary, and at that time consisted of but four wards, one of which was reserved for patients with diseases of the eye.

In 1933–1934 the new University Hospital was erected, and patients were admitted to this building in November 1934. The new hospital is situated at the southwest corner of Redwood and Greene Streets, and is consequently opposite the Medical School buildings. The students, therefore, are in close proximity and little time is lost in passing from the lecture halls and laboratories to the clinical facilities of the new building.

This new building, with its modern planning, makes a particularly attractive teaching hospital and is a very valuable addition to the clinical facilities of the Medical School.

The new hospital has a capacity of 435 beds and 50 bassinets devoted to general medicine, surgery, obstetrics, pediatrics, and the various medical and surgical specialties.

The teaching zone extends from the second to the eighth floor and comprises wards for surgery, medicine, obstetrics, pediatrics, and a large clinical lecture hall. There are approximately 270 beds available for teaching.

The space of the whole north wing of the second floor is occupied by the Department of Roentgenology. The east wing houses clinical pathology and special laboratories for clinical microscopy, biochemistry, bacteriology, and an especially well appointed laboratory for students' training. The south wing provides space for electro-cardiographic and basal metabolism departments, with new and very attractive air-conditioned or oxygen therapy cubicles. The west wing contains the Departments of Rhinolaryngology and Bronchoscopy, Industrial Surgery, Ophthalmology, and Male and Female Cystoscopy.

The third and fourth floors each provide two medical and two surgical wards. The fifth floor contains two wards for pediatrics, and on the sixth floor there are two wards for obstetrics. Each ward occupies the space of one wing of the hospital.

On the seventh floor is the general operating suite, the delivery suite, and the central supply station. The eighth floor is essentially a students' floor and affords a mezzanine over the operating and delivery suites, and a students' entrance to the clinical lecture hall.

In the basement there is a very well appointed Pathological Department with a large teaching autopsy room and its adjunct service of instruction of students in pathological anatomy.

The hospital receives a large number of accident patients because of its proximity to the largest manufacturing and shipping districts of the city.

The obstetrical service is particularly well arranged and provides accommodation for forty ward patients. This service, combined with an extensive home service, assures the student of abundant obstetrical training.

During the year ending December 31, 1939, 1224 cases were delivered in the hospital and 884 cases in the outdoor department. Students in the graduating class observed at least thirty-five cases, each student being required to deliver at least ten patients in their homes.

The dispensaries associated with the University Hospital and the Mercy Hospital are organized upon a uniform plan in order that the teaching may be the same in each. Each dispensary has the following departments: Medicine, Surgery, Pediatrics, Ophthalmology, Otology, Genito-Urinary, Gynecology, Gastro-Enterology, Neurology, Orthopaedics, Proctology, Dermatology, Laryngology, Rhinology, Cardiology, Tuberculosis, Psychiatry, Oral Surgery and Oncology.

All students in their junior year work each day during one-third of the year in the Departments of Medicine and Surgery of the dispensaries. In their senior year, all students work one hour each day in the special departments.

MERCY HOSPITAL

The Sisters of Mercy first assumed charge of the Hospital at the corner of Calvert and Saratoga Streets, then owned by the Washington University, in 1874. By the merger of 1878 the Hospital came under the control of the College of Physicians and Surgeons, but the Sisters continued their work of ministering to the patients.

In a very few years it became apparent that the City Hospital, as it was then called, was much too small to accommodate the rapidly growing demands upon it. However, it was not until 1888 that the Sisters of Mercy, with the assistance of the Faculty of the College of Physicians and Surgeons, were able to lay the cornerstone of the present Hospital. This building was completed and occupied late in 1889. Since then the growing demands for more space have compelled the erection of additions, until now there are accommodations for 310 patients.

In 1909 the name was changed from The Baltimore City Hospital to Mercy Hospital.

The clinical material in the free wards is under the exclusive control of the Faculty of the University of Maryland School of Medicine and College of Physicians and Surgeons.

The close union of the Hospital and College buildings greatly facilitates the clinical and didactic teaching.

THE BALTIMORE CITY HOSPITALS

The clinical facilities of the School of Medicine have been largely increased by the liberal decision of the Department of Public Welfare to allow the use of the wards of these hospitals for medical education. The autopsy material also is available for student instruction.

Members of the Junior Class make daily visits to these hospitals for clinical instruction in medicine, surgery, and the specialties.

The Baltimore City Hospitals consist of the following separate divisions:

The General Hospital, 400 beds, 60 bassinets.
The Hospital for Chronic Cases, 575 beds.
The Hospital for Tuberculosis, 280 beds.
The Psychopathic Hospital, 85 beds.
Infirmary (Home for Aged) 1053 beds.

THE JAMES LAWRENCE KERNAN HOSPITAL AND INDUSTRIAL SCHOOL OF MARYLAND FOR CRIPPLED CHILDREN

This institution is situated on an estate of 75 acres at Dickeyville. The site is just within the northwestern city limits and easy access of the city proper.

The location is ideal for the treatment of children, in that it affords all the advantages of sunshine and country air.

A hospital unit, complete in every respect, offers all modern facilities for the care of any orthopaedic condition in children.

The hospital is equipped with 100 beds—endowed, and city and state supported.

The Orthopaedic Dispensary at the University Hospital is maintained in closest affiliation and cares for the cases discharged from the Kernan Hospital. The Physical Therapy Department is very well equipped with modern apparatus and trained personnel. Occupational therapy has been fully established and developed under trained technicians.

THE BALTIMORE EYE, EAR, AND THROAT HOSPITAL

This institution was first organized and operated in 1882 as an outgrowth of the Baltimore Eye and Ear Dispensary, which closed on June 14, 1882. The name then given to the new hospital was The Baltimore Eye and Ear Charity Hospital. It was located at the address now known as 625 W. Franklin St. The out-patient department was opened on September 18th, 1882 and the hospital proper on November 1st of the same year. In 1898 a new building afforded 24 free beds and 8 private rooms; by 1907 the beds numbered 47; at present there are 60 beds, 29 of which are free. In 1922 the present hospital building at 1214 Eutaw Place was secured and in 1926 the dispensary was opened. In 1928 a clinical laboratory was installed. During 1939 the out-patient visits numbered 24,236.

Through the kindness of the Hospital's Board and Staff, our junior students have access to the dispensary which they visit in small groups for instruction in ophthalmology.

REQUIREMENTS FOR ADMISSION

METHOD OF MAKING APPLICATION

Application forms may be filed beginning October 1 for the following September classes. These forms may be secured from the Committee on Admissions, School of Medicine, University of Maryland, Baltimore.

APPLICATION FOR ADMISSION TO THE FIRST YEAR

Application for admission is made by filing the required form and by having all pertinent data sent directly to the Committee on Admissions, in accordance with the instructions accompanying the application.

APPLICATION FOR ADMISSION TO ADVANCED STANDING

Students who have attended approved medical schools are eligible to file applications for admission to the second- and third-year classes. These applicants must be prepared to meet the current first-year entrance requirements in addition to presenting acceptable medical school credentials, and a medical school record based on courses which are quantitatively and qualitatively equivalent to similar courses in this school.

Application to advanced standing is made in accordance with the instructions accompanying the application form.

MINIMUM REQUIREMENTS FOR ADMISSION

The minimum requirements for admission to the School of Medicine are:

(a) Graduation from an approved secondary school, or the equivalent in entrance examinations, and

(b) Three calendar years of acceptable premedical credit earned in an approved college of arts and sciences. The quantity and quality of this preprofessional course of study shall be not less than that required for recommendation by the institution where the premedical courses are being, or have been, studied.

The premedical curriculum shall include basic courses in

English
Biology
Inorganic Chemistry
Organic Chemistry
Physics
French or German

and such elective courses as will complete a balanced three-year schedule of study.

The elective courses should be selected from the following three groups:

Humanities

English (an advanced course in English composition should be taken, if possible)
Scientific German or French (A reading knowledge of either language is desirable, although German is preferred)
Philosophy

Natural Sciences

Comparative Vertebrate Anatomy
Embryology
Physical Chemistry or Quantitative Analysis
Mathematics

Social Sciences

Economics
History
Political Science
Psychology (a basic course should be taken)
Sociology, etc.

Careful attention should be given to the selection of elective courses in the natural sciences. Accordingly, it is suggested that the elective list given above be a guide in this connection and that the remainder of the college credits be accumulated from courses designed to promote a broad cultural development. Students should avoid the inclusion of college courses in subjects that occur in the medical curriculum, for example, histology, histological technique, human anatomy, bacteriology, physiology, neurology, physiological chemistry, etc.

It is not intended that these suggestions be interpreted to restrict the education of students who exhibit an aptitude for the natural sciences or to limit the development of students who plan to follow research work in the field of medicine.

In accepting candidates for admission, preference will be given to those applicants who have acceptable scholastic records in secondary school and college, satisfactory scores in the Medical Aptitude Test, favorable letters of recommendation from their premedical committees, or from one instructor in each of the departments of biology, chemistry, and physics, and who in all other respects give every promise of becoming successful students and physicians of high standing.

Those candidates for admission who are accepted will receive certificates of entrance from the Director of Admissions of the University.

COMBINED COURSE IN ARTS AND SCIENCES, AND MEDICINE

A combined seven years' curriculum leading to the degrees of Bachelor of Science and Doctor of Medicine is offered by the University of Maryland. The first three years are taken in residence in the College of Arts and Sciences at College Park, and the last four years in the School of Medicine in Baltimore. (See University catalogue for details of quantitative and qualitative premedical course requirements.)

Upon the successful completion of the first year in the School of Medicine, and upon the recommendation of the Dean, the degree of Bachelor of

Science may be conferred by the College of Arts and Sciences at the Commencement following the *second year* of residence.

STATE MEDICAL STUDENT QUALIFYING CERTIFICATES

Candidates for admission who live in or expect to practice medicine in Pennsylvania, New Jersey or New York, should apply to their respective state boards of education for medical student qualifying certificates (Pennsylvania and New Jersey) or approval of applications for medical student qualifying certificates (New York).

Those students who are accepted must file satisfactory State certificates in the office of the Committee on Admissions, School of Medicine, before registration. No exceptions will be made to this requirement.

Addresses of the Respective State Boards:

Director of Credentials Section, Pennsylvania Department of Public Instruction, Harrisburg, Pa.

Chief of the Bureau of Credentials, New Jersey Department of Public Instruction, Trenton, N. J.

Supervisor of Qualifying Certificates, The State Education Department, Examinations and Inspections Division, Albany, N. Y.

DEFINITION OF RESIDENCE STATUS OF STUDENTS*

Students who are minors are considered to be resident students if, at the time of their registration, the parents* have been residents of this State for at least one year.

Adult students are considered to be resident students if, at the time of their registration, they have been residents of this State for at least one year, provided such residence has not been acquired while attending any school or college in Maryland.

The status of the residence of a student is determined at the time of his first registration in the University and may not thereafter be changed by him unless, in the case of a minor, his parents* move to and become legal residents of this State by maintaining such residence for at least one full calendar year. However, the right of the student (minor) to change from a non-resident to a resident status must be established by him prior to registration for a semester in any academic year.

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* The term "parents" includes persons who, by reason of death or other unusual circumstances, have been legally constituted the guardians of or stand in loco parentis to such minor students.

FEES

Matriculation fee (paid once)	$10.00
Tuition fee (each year)—Residents of Maryland	450.00
Tuition fee (each year)—Non-Residents	600.00
Laboratory fee (each year)	25.00
Conditioned examination fee (each subject)	5.00
Student health service fee (each year)	10.00
Student activities fee (each year)	5.00
Maintenance and service fee (each year):	
First year	6.00
Second year	3.00
Third year	2.30
Fourth year	7.00
Graduation fee	15.00
Transcript fee to graduates. First copy gratis, thereafter, each copy	1.00

RULES FOR PAYMENT OF FEES

No fees are returnable.

The above fees apply to all students who matriculate in the School of Medicine in any class for the session beginning September 26, 1940.

Make all checks or money orders payable to the University of Maryland.

When offering checks or money orders in payment of tuition and other fees, students are requested to have them drawn in the exact amount of such fees. Personal checks whose face value is in excess of the fees due will be accepted for collection only.

Acceptance.—The matriculation fee of $10.00 is payable on acceptance. In addition, a deposit on tuition of $50.00 is required of accepted applicants before the expiration date specified in the offer of acceptance. This $60.00 deposit is not returnable and will be forfeited if the applicant fails to register, or it will be applied to the applicant's first semester's charges on registration.

Registration.—All students, after proper certification, are required to register at the Office of the Registrar. (See calendar in front of this bulletin for dates for the payments of fees, and the note regarding late registration fee.)

One-half of the tuition fee, the laboratory fee, the student health fee, the student activities fee, and the maintenance and service fee are payable on the date specified for registration for the first semester.

The remainder of the tuition fee shall be paid on the date of registration for the second semester. Fourth year students shall pay the graduation fee, in addition, at this registration.

PENALTY FOR NON-PAYMENT OF FEES

If fees are not paid on the specified registration dates, a penalty of $5.00 will be added.

If a satisfactory settlement, or an agreement for settlement, is not made with the Dean within ten days after a payment is due, the student automatically is debarred from attendance on classes and will forfeit the other privileges of the School of Medicine.

CONDITIONED EXAMINATION FEE

A student who is eligible to conditioned examinations must notify the Dean two weeks in advance of his intention to take the examinations. He will pay the Comptroller $5.00 for each subject in which he is to be examined, and he must present the receipt to the Dean for endorsement before he will be permitted to take the examinations.

MAINTENANCE AND SERVICE FEE

This fee pays for maintenance of loan collections, provides for photograph requests, and pays room-rent for the third- and fourth-year students when on outside obstetrics. The charges are as follows:

First year. Loan collections in Anatomy ($3.00), Histology & Neuro-Anatomy ($2.00), and photographs ($1.00).

Second year. Loan collection in Pathology ($2.00) and photographs ($1.00).

Third year. Room-rent for obstetrics at City Hospitals ($1.30) and photographs ($1.00).

Fourth year. Room-rent while on outside obstetrics at University Hospital ($6.00) and photographs ($1.00).

Fourth-year students who desire to work during the summer in outside obstetrics will be required to pay the $6.00 fee for room-rent when they register for this course.

PERSONAL EXPENSES

The following estimates of personal expenses for the academic year of eight months have been prepared by students, and are based upon actual experience. In addition to these the student must bear in mind the expenditure for a microscope.

	Low	*Average*	*Liberal*
Books	$50	$75	$100
College Incidentals	20	20	20
Board, eight months	200	250	275
Room rent	64	80	100
Clothing and laundry	50	80	150
All other expenses	25	50	75
Total	$409	$556	$720

STUDENT HEALTH SERVICE

The Medical Council has made provision for the systematic care of students in the Medical School, according to the following plan:

1. *Preliminary Examination*—All new students will be examined during the first week of the semester. Notice of the date, time, and place of the examination will be announced to the classes and on the bulletin board. The passing of this physical examination is necessary before final acceptance of any student.

2. *Medical Attention*—Students in need of medical attention will be seen by the School Physician, Dr. T. N. Carey, in his office at the Medical School, between 4 and 5 P.M., daily, except Saturday and Sunday. In cases of necessity, students will be seen at their homes.

3. *Hospitalization*—If it becomes necessary for any student to enter the hospital during the school year, the Medical Council has arranged for the payment of part or all of his hospital expenses, depending on the length of his stay and special expenses incurred. This applies only to students admitted through the School Physician's Office.

4. *Physical Defects*—Prospective students are advised to have any known physical defects corrected before entering school in order to prevent loss of time which later correction might incur.

All students are urgently advised to undergo an eye examination at the hands of an oculist (Doctor of Medicine) before entering the School of Medicine. In many students long study hours bring out unsuspected eye defects which cause much loss of time and inefficiency in study if not corrected until after school work is under way.

An eye examination will be required beginning September, 1941.

5. *Limitations*—It is not the function of this service to treat chronic conditions contracted by students before admission, nor to extend treatment to acute conditions arising in the period between academic years, unless the School Physician recommends this service.

GENERAL RULES

All rules, as well as fees, relate to the year ending June 7, 1941 only. The right is reserved to make changes in the curriculum, the requirements for graduation, the fees and in any of the regulations whenever the University authorities deem it expedient.

ADVANCEMENT AND GRADUATION

1. All students are required to take the spring examinations unless excused by the Dean.

2. No student will be permitted to advance from a lower to a higher class with conditions.

3. Should a student be required to repeat any year in the course, he must pay regular fees.

4. A student failing in final examinations for graduation at the end of the

fourth year will be required to repeat the entire course of the fourth year and to take examinations in such other branches as may be required, should he again be permitted to enter the school as a candidate for graduation.

5. The general fitness of a candidate for graduation, as well as the results of his examinations, will be taken into consideration by the Faculty.

EQUIPMENT

6. At the beginning of the first year, all students must be prepared to purchase microscopes of a satisfactory type equipped with a mechanical stage and a sub-stage lamp. The cost is about $157.00, when new.

A standard microscope of either Bausch & Lomb, Leitz, Spencer, or Zeiss make, fitted with the following attachments, will fill the requirements:

Triple nose piece: 10 x and 5 x Oculars
Wide aperture stage: 16 mm. and 4 mm. Objectives
Quick Screw condenser (Abbe): 1.9 mm. 125 N.A. Oil Immersion Lens

All used microscopes are subject to inspection and approval before their use in the laboratory is permitted. The student is cautioned against the purchase of such an instrument before its official approval by the school.

7. Students in the third- and fourth-year classes are required to furnish their own ophthalmoscopes.

8. Third-year students are required to purchase haemocytometers.

STATE QUALIFYING CERTIFICATES

9. Candidates for admission who live in or expect to practice medicine in Pennsylvania, New Jersey or New York must file State certificates in the office of the Committee on Admissions, School of Medicine, before registration. No exception will be made to this rule.

EYE EXAMINATION BEFORE ADMISSION

(*Effective September, 1941*)

10. Each new matriculate in each class is required to present to the Committee on Admissions a certificate from an oculist, (a graduate in medicine) that the matriculate's eyes have been examined and are in condition, with or without glasses as the case may be, to endure the strain of close and intensive reading.

It is required that this examination be completed within three months prior to registration and that the certificate be mailed to the Committee on Admissions not later than one month before registration.

AWARDING OF COMBINED DEGREES

11. Students entering the School of Medicine on a three-year requirement basis from colleges which usually grant a degree on the

successful completion of the first year of medicine, are restricted by the following rule:

> Upon successful completion of the first year in the School of Medicine, and upon recommendation of the Dean of the School of Medicine, the degree of Bachelor of Science may be conferred by a College of Arts and Sciences following the student's *second year* of residence in the School of Medicine.

COST OF TRANSCRIPTS

12. Graduates will receive the first transcript of record without charge. Subsequent copies will cost one dollar each.

PRIZES

THE FACULTY PRIZE

The Faculty each year awards a Gold Medal to the Graduate who during the four years of his course has shown the greatest proficiency in preparing for the practice of medicine. The five candidates standing next in order will be awarded Certificates of Honor.

THE DR. A. BRADLEY GAITHER MEMORIAL PRIZE

A prize of $25.00 is given each year by Mrs. A. Bradley Gaither as a memorial to the late Dr. A. Bradley Gaither, to the student in the senior class doing the best work in Genito-Urinary Surgery.

THE SAMUEL M. SHOEMAKER PRIZE

An annual prize of $25.00 has been established by Mrs. Samuel M. Shoemaker and Mrs. Bartlett F. Johnston as a memorial to Samuel M. Shoemaker for the best essay on "Milk in Relation to Public Health" written by a student in the senior class.

SCHOLARSHIPS

All scholarships are assigned for one academic year, unless specifically reawarded on consideration of an application.

Official application forms are obtainable at the Dean's office, where they should be filed before July 1, for the ensuing academic year.

THE DR. SAMUEL LEON FRANK SCHOLARSHIP

(Value $100.00)

This scholarship was established by Mrs. Bertha Rayner Frank as a memorial to the late Dr. Samuel Leon Frank, an alumnus of this University.

It is awarded by the Trustees of the Endowment Fund of the University each year upon nomination by the Medical Council "to a medical student of the University of Maryland, who in the judgment of said Council, is of good character and in need of pecuniary assistance to continue his medical course."

This scholarship is awarded to a second, third or fourth year student who has successfully completed one year's work in this school. No student may hold this scholarship for more than two years.

THE CHARLES M. HITCHCOCK SCHOLARSHIPS

(Value $100.00 each)

Two scholarships were established from a bequest to the School of Medicine by the late Charles M. Hitchcock, M.D., an alumnus of the University.

These scholarships are awarded annually by the Trustees of the Endowment Fund of the University, upon nomination by the Medical Council, to students who have meritoriously completed the work of at least the first year of the course in medicine, and who present to the Council satisfactory evidence of a good moral character and of inability to continue the course without pecuniary assistance.

THE RANDOLPH WINSLOW SCHOLARSHIP

(Value $100.00)

This scholarship was established by the late Randolph Winslow, M.D., LL.D.

It is awarded annually by the Trustees of the Endowment Fund of the University, upon nomination by the Medical Council, to a "needy student of the Senior, Junior, or Sophomore Class of the Medical School."

"He must have maintained an average grade of 85% in all his work up to the time of awarding the scholarship."

"He must be a person of good character and must satisfy the Medical Council that he is worthy of and in need of assistance."

THE DR. LEO KARLINSKY MEMORIAL SCHOLARSHIP

(Value $125.00)

This scholarship was established by Mrs. Ray Mintz Karlinsky as a memorial to her husband, the late Dr. Leo Karlinsky, an alumnus of the University.

It is awarded annually by the Trustees of the Endowment Fund of the University, upon the nomination of the Medical Council, to "a needy student of the Senior, Junior or Sophomore Class of the Medical School."

He must have maintained in all his work up to the time of awarding the scholarship a satisfactory grade of scholarship.

He must be a person of good character and must satisfy the Medical Council that he is worthy of and in need of assistance.

THE UNIVERSITY SCHOLARSHIP

A scholarship which entitles the holder to exemption from payment of tuition fee for the year, is awarded annually by the Medical Council to a student of the senior class in need of assistance who presents to the Medical Council satisfactory evidence of good character and scholarship.

THE FREDERICA GEHRMANN SCHOLARSHIP

(Value $200.00)

(Not open to holders of Warfield and Cohen Scholarships)

This scholarship was established by the bequest of the late Mrs. Frederica Gehrmann and is awarded to a third-year student who at the end of the second year has passed the best practical examinations in Physiology, Pharmacology, Pathology, Bacteriology, Immunology, Serology, Surgical Anatomy and Neuro-Anatomy.

THE CLARENCE AND GENEVRA WARFIELD SCHOLARSHIPS

(Value $300.00 each)

There are five scholarships established by the Regents from the income of the fund bequeathed by the will of Dr. Clarence Warfield.

Terms and Conditions: These scholarships are available to students of any of the classes of the course in medicine. Preference is given to students from the counties of the State of Maryland which the Medical Council may from time to time determine to be most in need of medical practitioners.

Any student receiving one of these scholarships must agree, after graduation and a year's internship, to undertake the practice of medicine, for a term of two years, in the county to which the student is accredited, or in a county selected by the Council. In the event that a student is not able to comply with the condition requiring him to practice in the county to which he is accredited by the Council, the money advanced by the Regents shall be refunded by the student.

THE ISRAEL AND CECELIA E. COHEN SCHOLARSHIP

(Value $150.00)

This scholarship was established by the late Eleanor S. Cohen in memory of her parents, Israel and Cecelia E. Cohen. Terms and conditions: This scholarship will be available to students of any one of the classes of the course in Medicine; preference is given to students of the counties in the State of Maryland which the Medical Council may from time to time determine to be most in need of medical practitioners. Any student receiving one of these scholarships must, after graduation and a year's internship, agree to undertake the practice of medicine for a term of two years in the county to which the student is accredited, or in a county selected by the Council. In the event that a student is not able to comply with the condition requiring him to practice in the county to which he is accredited by the Council, the money advanced by the Regents shall be refunded.

THE DR. HORACE BRUCE HETRICK SCHOLARSHIP

(Value $125.00)

This scholarship was established by Dr. Horace Bruce Hetrick as a memorial to his sons, Bruce Hayward Hetrick and Augustus Christian Hetrick. It is to be awarded by the Medical Council to a student of the senior class.

THE MEDICAL ALUMNI ASSOCIATION SCHOLARSHIP

(Value $300.00)

This scholarship was established by The Medical Alumni Association of the University of Maryland.

It is awarded, on recommendations of the Board of Directors of the Medical Alumni Association and of the Chairman of the Committee on Admissions of the School of Medicine, to a student of the first year who must furnish satisfactory evidence of good character and scholastic accomplishment and of his inability to pursue the medical course without pecuniary support.

ORGANIZATION OF THE CURRICULUM

The following curriculum is the result of a thorough revision of teaching in this school in order to meet modern requirements. The multiplication of specialties in medicine and surgery necessitates a very crowded course and the introduction of electives will very soon be depended on to solve some of the difficulties.

The curriculum is organized under eleven departments.

1. Anatomy (including Histology and Embryology).
2. Physiology.
3. Bacteriology and Immunology.
4. Biological Chemistry.
5. Pharmacology and Materia Medica.
6. Pathology.
7. Medicine (including Medical Specialties).
8. Surgery (including Surgical Specialties).
9. Obstetrics.
10. Gynecology.
11. Ophthalmology.

The instruction is given in four years of graded work.

Several courses of study extend through two years or more, but in no case are the students of different years thrown together in the same course of teaching.

The first and second years are devoted largely to the study of the structures and functions of the normal body. Laboratory work occupies most of the student's time during these two years.

Some introductory instruction in Medicine and Surgery is given in the second year. The third and fourth years are almost entirely clinical.

A special feature of instruction in the school is the attempt to bring together teacher and student in close personal relationship. In many courses of instruction the classes are divided into small groups and a large number of instructors insures attention to the requirements of each student.

In most courses the final examination as the sole test of proficiency has disappeared and the student's final grade is determined largely by partial examinations, recitations and assigned work carried on throughout the course.

DEPARTMENT OF GROSS ANATOMY

Eduard Uhlenhuth	Professor of Anatomy
[1]Frank H. J. Figge	Associate Professor of Gross Anatomy
Monte Edwards	Associate Professor of Surgery
James C. Lipsett	Assistant Professor of Gross Anatomy
*E. Eugene Covington	Associate in Gross Anatomy
Harry C. Hull	Instructor in Surgery
Otto C. Brantigan	Instructor in Gross Anatomy
Harry S. Shelley	Instructor in Gross Anatomy
†James C. Plagge	Instructor in Gross Anatomy
†James U. Thompson	Instructor in Gross Anatomy
Thurston R. Adams	Assistant in Surgery
‡Karl F. Mech	Assistant in Gross Anatomy

Gross Anatomy. *First year.* Four lectures and conferences and nineteen hours of laboratory (dissection of the human subject) are given each week during the first semester. Drs. Uhlenhuth, Figge, Lipsett, Covington, Brantigan, Shelley and Mech.

Total hours: 350.

Topographic and Surgical Anatomy. *Second year.* The course is designed to bridge the gap between anatomy in the abstract and clinical anatomy as applied to the study and practice of medicine and surgery.

The teaching is done in the anatomical laboratory, and students are required to demonstrate all points, outlines, and regions on the cadaver. Underlying regions are dissected to bring out outlines and relations of structures.

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[1] On leave 1940–1941.

Two lectures and two laboratory periods are given each week during the second semester. Drs. Monte Edwards, Hull, Brantigan and Adams.

Total hours: 96.

Advanced Anatomy (*elective course*). Selected problems in gross anatomy. This course is intended primarily to offer to the sophomore student an opportunity of extending the knowledge secured in the freshman course. Drs. Uhlenhuth, Figge, Edwards, Lipsett and Brantigan.

Total hours: 64.

In addition to the above courses, facilities for special anatomical problems are offered to the more advanced student and physician.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF HISTOLOGY AND EMBRYOLOGY

Carl L. Davis	Professor of Anatomy
O. G. Harne	Associate Professor of Histology
John F. Lutz	Assistant Professor of Histology
Joseph Pokorny	Instructor in Histology
Martin J. Hanna	Instructor in Histology

First Year. The course in histology is divided equally between the study of the fundamental tissues and that of organs. Throughout the entire course the embryology of the part being studied precedes the study of the fully developed tissue. Thus embryology becomes a correlated part of the whole subject of microscopic anatomy and not an independent subject.

Each student is furnished a set of histological slides, previously prepared in our own laboratory, thus insuring a uniform and satisfactory quality of material for study and permitting the time of the student to be expended in the study of material rather than in the technic of its preparation. The object of the course is to present the evidence of function as shown by the structure of tissues and organs. Drs. Davis, Lutz and Harne.

An optional laboratory course is offered. This supplements the required course giving laboratory experience which can not be incorporated in the former. No added charge is made for the course.

Total hours: 150.

NEURO-ANATOMY

First Year. Neuro-anatomy embraces a study of the fundamental structure of the central nervous system as applied to its function. An abundance of material permits of individual dissection of the human brain. A series of appropriately stained sections of the human brain stem is furnished each student for the microscopic study of the internal structure of the nervous system. Dr. Davis and Dr. Lutz.

Total hours: 100.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PHYSIOLOGY

William R. Amberson	Professor of Physiology
Dietrich Conrad Smith	Associate Professor of Physiology
Robert H. Oster	Assistant Professor of Physiology
‡Edward G. Boettiger	Instructor in Physiology
†Walter H. Gerwig, Jr.	Instructor in Physiology
Joye E. Jacobs	Assistant in Physiology
J. Victor Monke	Weaver Fellow in Physiology

Four lectures, two conferences and two laboratory periods a week, September to January inclusive. The fundamental concepts of physiology are presented with special reference to mammalian problems.

Total hours: 224.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF BACTERIOLOGY AND IMMUNOLOGY

Frank W. Hachtel	Professor of Bacteriology
James G. McAlpine	Associate Professor of Bacteriology
J. A. F. Pfeiffer	Instructor in Bacteriology
Henry F. Buettner	Instructor in Bacteriology
H. Edmund Levin	Instructor in Bacteriology

The principles of general bacteriology are taught by quiz, conference, and lecture.

Instruction in bacteriology is given in the laboratory to students of the second year class during the first semester. This includes the methods of preparation of culture media, the study of pathogenic bacteria, and the bacteriological examination of water and milk. The bacteriological diagnosis of communicable diseases is also included.

The principles of immunology are presented by means of quizzes, conferences, and lectures to the second year class in the second semester.

The course includes a consideration of infection and immunity, the nature and action of the various antibodies, complement fixation and flocculation tests, hypersensitiveness, and the preparation of bacterial vaccines.

Experiments are carried out by the class in the laboratory. During the latter half of the semester the class is divided into sections.

Total hours: Bacteriology 120.
Immunology 72.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF BIOLOGICAL CHEMISTRY

H. Boyd Wylie	Professor of Biological Chemistry
Emil G. Schmidt	Associate Professor of Biological Chemistry
Frank N. Ogden	Associate in Biological Chemistry
Dorothy E. Schmalzer	Assistant in Biological Chemistry
Howard B. Mays	Fellow in Biological Chemistry
————————	Research Assistant in Biological Chemistry

This course is designed to present the principles of biological chemistry and to indicate their applications to the clinical aspects of medicine. The phenomena of living matter and its chief ingredients, secretions and excretions are discussed in lectures and conferences and examined experimentally. Training is given in current biochemical methods of investigation. Total hours: 208.

Graduate Courses. Consult the catalogue of the Graduate School for descriptions of the graduate courses offered by members of the staff.

DEPARTMENT OF PHARMACOLOGY

John C. Krantz, Jr.	Professor of Pharmacology
C. Jelleff Carr	Associate Professor of Pharmacology
William Ellsworth Evans	Assistant Professor of Pharmacology
Ruth Musser	Instructor in Pharmacology
Marius P. Johnson	Assistant in Pharmacology
Frederick K. Bell	U. S. Pharmacopoeia Fellow
Frances F. Beck	International Cancer Foundation Fellow
Sylvan Forman	Isaac E. Emerson Fellow in Pharmacology
Fred W. Ellis	Fellow in Pharmacology
†Wilson C. Grant, M.S.	Research Assistant in Pharmacology, International Cancer Research Foundation
†Nellie Cone, A.B.	Emerson Fellow in Pharmacology
†Edward Merdinyan, M.S.	Emerson Fellow in Pharmacology

This course is designed to include those phases of pharmacology necessary for an intelligent use of drugs in the treatment of disease. The didactic instruction includes materia medica, pharmacy, prescription-writing, toxicology, posology, pharmacodynamics, and experimental therapeutics. The laboratory exercises parallel the course of lectures.

In addition, optional conference periods and lectures are available for students desiring further instruction or advice.

Total hours: 176.

For a description of the graduate courses offered by the members of the staff, consult the catalogue of the Graduate School.

DEPARTMENT OF PATHOLOGY

Hugh R. Spencer	Professor of Pathology
Robert B. Wright	Assistant Professor of Pathology
C. Gardner Warner	Assistant Professor of Pathology
Walter C. Merkel	Assistant Professor of Pathology
Howard J. Maldeis	Associate in Pathology
Albert E. Goldstein	Associate in Pathology
Frank B. Kindell	Associate in Pathology
Wm. S. Love, Jr.	Instructor in Pathology
Leon Freedom	Instructor in Pathology
Benjamin Abeshouse	Instructor in Pathology
Milton S. Sacks	Instructor in Pathology
D. James Greiner	Instructor in Pathology
Conrad B. Acton	Assistant in Pathology
John E. Savage	Assistant in Pathology
Harry C. Hull	Assistant in Pathology
Henry L. Wollenweber	Assistant in Pathology
‡Roland E. Bieren	Assistant in Pathology
John L. Atkins	Weaver Fellow in Pathology
†Ephraim T. Lisansky	Weaver Fellow in Pathology

Courses of instruction in Pathology are given during the second and third years. The courses are based on the previous study of normal structure and function and aim to outline the history of disease. The relationship between clinical symptoms and anatomical lesions is constantly stressed.

General Pathology. (*Second Semester, Second Year.*) This course includes the study of disturbances of the body fluids; disturbances of structure, nutrition and metabolism of cells; disturbances of fat, carbohydrate and protein metabolism; disturbances of pigment metabolism; inflammation and tumors.

Laboratory instruction is based on the study of prepared slides (loan collection) and corresponding gross material.

Applied Pathology, Including Gross Morbid Anatomy and Morbid Physiology. (*Third Year.*) The laboratory instruction in this course is carried out in small teaching museums where prepared specimens and material from autopsies with clinical histories and sections are available for study. For this work the class is divided into small groups. Clinical correlation is stressed.

Autopsies. (*Third Year.*) Students in small groups attend autopsies at the morgues of the University Hospital and the Baltimore City Hospitals.

Clinical Pathological Conference. (*Fourth Year.*) These exercises are held in collaboration with the Department of Medicine. Selected cases are discussed and autopsy findings are presented.

Second year	184 hours
Third year	160 hours
Fourth year	30 hours
Total	374 hours

DEPARTMENT OF MEDICINE

Maurice C. Pincoffs	Professor of Medicine
Jos. E. Gichner	Professor of Clinical Medicine and Physical Therapeutics
G. Carroll Lockard	Professor of Clinical Medicine
Harvey G. Beck	Professor of Clinical Medicine
Harry M. Stein	Professor of Clinical Medicine
Thomas R. Sprunt	Professor of Clinical Medicine
Paul W. Clough	Associate Professor of Medicine
Sydney R. Miller	Associate Professor of Medicine
Walter A. Baetjer	Associate Professor of Medicine
Wm. S. Love, Jr.	Associate Professor of Medicine
Raymond Hussey	Associate Professor of Medicine
L. A. M. Krause	Associate Professor of Medicine
H. R. Peters	Associate Professor of Medicine
T. Nelson Carey	Associate Professor of Medicine
William H. Smith	Associate Professor of Clinical Medicine
H. J. Maldeis	Associate Professor of Medical Jurisprudence
George McLean	Assistant Professor of Medicine
John E. Legge	Assistant Professor of Medicine
Thomas C. Wolff	Assistant Professor of Medicine
H. M. Bubert	Assistant Professor of Medicine
E. B. Freeman	Lecturer in Medicine
Charles R. Goldsborough	Lecturer in Medicine
W. N. Bispham, Col., M. C., U. S. A. (ret.)	Lecturer in Medicine
J. S. Eastland	Associate in Medicine
Wetherbee Fort	Associate in Medicine
Frank J. Geraghty	Associate in Medicine
L. P. Gundry	Associate in Medicine
E. H. Tonolla	Associate in Medicine
†Milton S. Sacks	Associate in Medicine
Samuel T. Helms	Instructor in Medicine
Earl L. Chambers	Instructor in Medicine
David Tenner	Instructor in Medicine
R. B. Mitchell, Jr.	Instructor in Medicine
Samuel Legum	Instructor in Medicine
Harry V. Langeluttig	Instructor in Medicine
Robert W. Garis	Instructor in Medicine
M. S. Shiling	Instructor in Medicine
Sol Smith	Instructor in Medicine
H. Edmund Levin	Instructor in Medicine
Robert A. Reiter	Instructor in Medicine
W. Grafton Hersperger	Instructor in Medicine

Meyer W. Jacobson	Instructor in Medicine
Conrad Acton	Instructor in Medicine
Hugh G. Whitehead	Instructor in Medicine
Philip D. Flynn	Instructor in Medicine
*Edward S. Kallins	Instructor in Medicine
†Irving Freeman	Instructor in Medicine
W. H. Triplett	Assistant in Medicine
Morris Fine	Assistant in Medicine
William H. Grenzer	Assistant in Medicine
George Silverton	Assistant in Medicine
Lawrence Katzenstein	Assistant in Medicine
Leon Ashman	Assistant in Medicine
Harry M. Robinson, Jr.	Assistant in Medicine
Nathaniel Beck	Assistant in Medicine
Joseph M. Blumberg	Assistant in Medicine
John A. Myers	Assistant in Medicine
Kurt Levy	Assistant in Medicine
Paul E. Carliner	Assistant in Medicine
Samuel Snyder	Assistant in Medicine
‡Edward F. Cotter	Assistant in Medicine
‡S. Edwin Muller	Assistant in Medicine
William G. Helfrich	Assistant in Medicine
Louis J. Kroll	Assistant in Medicine
†Frederick J. Vollmer	Assistant in Medicine
†John L. Atkins	Assistant in Medicine
William K. Waller	Clinical Fellow in Medicine

GENERAL OUTLINE

Second Year

Introduction to clinical medicine.

- (a) Introductory physical diagnosis.
  (1 hour a week, first semester; 2 hours a week, second semester.)
- (b) Medical clinics.
  (1 hour a week, second semester.)

Third Year

I. The methods of examination (13 hours a week).
  - (a) History taking.
  - (b) Physical diagnosis.
  - (c) Clinical pathology.

These subjects are taught and practiced in the hospital out-patient department and in the clinical laboratory.

II. The principles of medicine (200 hours).
  - (a) Lectures, clinics and demonstrations in general medicine, neurology, pediatrics, psychiatry and preventive medicine.

III. The principles of therapeutics (15 hours).
  Lectures and demonstrations.

Fourth Year

The practice of medicine.

I. Clinical clerkship on the medical wards.
  (26 hours a week for ten weeks.)

(a) Responsibility, under supervision, for the history, physical examination, laboratory examinations and progress notes of assigned cases.
(b) Ward classes in general medicine, the medical specialties, and therapeutics.

II. Clinics in general medicine and the medical specialties.
(6 hours a week.)
III. Dispensary work in the medical specialties.
IV. Clinical pathological conferences (1 hour a week).

MEDICAL DISPENSARY WORK

The medical dispensaries of both the Mercy and the University Hospitals are utilized for teaching in the third year. Each student spends two hours daily for ten weeks in dispensary work. The work is done in groups of four to six students under an instructor. Systematic history-taking is especially stressed. Physical findings are demonstrated. The student becomes familiar with the commoner acute and chronic disease processes.

PHYSICAL DIAGNOSIS

Second Year. Didactic lectures and practical demonstrations in topographical anatomy and normal physical signs.

Third Year. The class is divided into small groups, and each section receives instruction for two hours daily for ten weeks. This course is given at the City Hospitals. The large clinical material there is utilized to give each student the opportunity to familiarize himself with the common types of bodily structure, with the normal variations in physical signs and with the physical signs of the chief pulmonary, circulatory and abdominal diseases.

A course of lectures (1 hour a week) on physical diagnosis supplements the practical work in this subject.

THERAPEUTICS

Third Year. General therapeutics and materia medica are taken up and an effort is made to familiarize the student with the practical treatment of disease. The special therapy of the chief diseases is then reviewed. One hour a week. Dr. Lockard.

Fourth Year. Special consideration is given to the practical application of therapeutic principles in bedside teaching and the chief therapeutic methods are demonstrated.

Students attend therapeutic ward rounds once a week throughout their medical trimester.

TUBERCULOSIS

During the third year in connection with the instruction in physical diagnosis a practical course is given at the Municipal Tuberculosis Hospital. Stress is laid upon the recognition of the physical signs of the disease, as well as upon its symptomatology and gross pathology.

CARDIOLOGY

During the fourth year an elective course in cardiology is offered at the Mercy Hospital. The course occupies one and one-half hours weekly. Physical diagnosis, electrocardiography and the therapeutic management of cardiac cases are stressed.

SYPHILIS

Third Year. During the third year the subject of syphilis is dealt with in the lecture course.

Fourth Year. An elective course in the therapeutic management of syphilis is offered in the dispensary.

CLINICAL PATHOLOGY

†Milton S. Sacks	Associate in Medicine and Head of Department of Clinical Pathology
H. R. Peters	Associate Professor of Medicine
Samuel T. Helms	Instructor in Medicine
Hugh G. Whitehead	Assistant in Medicine
Joseph M. Blumberg	Assistant in Medicine
John A. Myers	Assistant in Medicine
William K. Waller	Clinical Fellow in Medicine

During the third year the student is thoroughly drilled in the technic of the usual clinical laboratory work, so that he is able to perform all routine examinations which may be called for during his fourth year, in connection with the work in the wards and dispensary.

The practical work is supplemented by a series of didactic lectures and demonstrations in which the entire teaching staff of the department takes an active part. The microscopical and chemical study of blood, exudates and transudates, gastric juice, spinal fluid, feces and urine are successively taken up, and special attention directed to the clinical significance of the findings.

Clinical parasitology from the standpoint of the infecting agent and the carrier is given careful consideration.

The entire course is thoroughly practical. Each student provides his own microscope and blood counters and is supplied a haemoglobinometer for his exclusive use. Every two students are equipped with a special laboratory outfit for all routine purposes.

During the fourth year the student applies in the laboratories of the various affiliated hospitals what he has learned during the preceding year.

He is also supplied with a laboratory outfit which is sufficiently complete to enable him to work independently of the general equipment. Special instructors are available during certain hours to give necessary assistance and advice.

GASTRO-ENTEROLOGY

Theodore H. Morrison	Clinical Professor of Gastro-Enterology
Maurice Feldman	Assistant Professor of Gastro-Enterology
Zachariah Morgan	Assistant Professor of Gastro-Enterology
Samuel Morrison	Assistant Professor of Gastro-Enterology
Joseph Sindler	Associate in Gastro-Enterology
Z. Vance Hooper	Associate in Gastro-Enterology
M. S. Koppelman	Instructor in Gastro-Enterology
Ernest Levi	Instructor in Gastro-Enterology
Albert J. Shochat	Instructor in Gastro-Enterology
John A. Myers	Assistant in Gastro-Enterology
William Greenfeld	Assistant in Gastro-Enterology
Nathaniel M. Beck	Assistant in Gastro-Enterology

Third Year. A series of six lectures is given on the diseases of the digestive tract.

Fourth Year. Clinics and demonstrations are given to the class for one hour a week; dispensary instruction to small groups throughout the entire session. Practical instruction is given in the use of modern methods of study of the diseases of the gastro-intestinal tract.

PSYCHIATRY

Ross McC. Chapman	Professor of Psychiatry
Ralph P. Truitt	Associate Professor of Psychiatry
Lawrence F. Woolley	Associate Professor of Psychiatry
Harry Goldsmith	Assistant Professor of Psychiatry
H. W. Newell	Assistant Professor of Psychiatry
Harry M. Murdock	Assistant Professor of Psychiatry
†Leslie B. Hohman	Lecturer in Psychiatry
A. Russell Anderson	Instructor in Psychiatry
†Philip S. Wagner	Instructor in Psychiatry
Gordon M. Stephens	Fellow in Psychiatry

First Year. The student attends six lectures dealing with the development and function of the normal personality.

Second Year. The student attends fourteen lectures dealing with psychopathology.

Third Year. Six lectures on psychopathology are given; twelve hours of lectures and demonstrations are devoted to reaction types; six hours of lectures and demonstrations to the psychoses; thirty hours to history-taking and the study of cases in the out-patient clinic.

Fourth Year. The neuroses, psychoneuroses and psychoses are dealt with in ten hours of lectures and demonstrations. In this year the class is divided into sections for clinical conferences on selected cases.

PEDIATRICS

C. Loring Joslin	Professor of Pediatrics
Edgar B. Friedenwald	Professor of Clinical Pediatrics
T. Campbell Goodwin	Associate Professor of Pediatrics
John H. Traband	Assistant Professor of Pediatrics
Albert Jaffe	Assistant Professor of Pediatrics
A. H. Finkelstein	Assistant Professor of Pediatrics
Frederick B. Smith	Assistant Professor of Pediatrics
J. Edmund Bradley	Assistant Professor of Pediatrics
William J. Todd	Associate in Pediatrics
Clewell Howell	Associate in Pediatrics
Samuel S. Glick	Associate in Pediatrics
F. Stratner Orem	Associate in Pediatrics
William M. Seabold	Associate in Pediatrics
*Thomas J. Coonan	Associate in Pediatrics
M. Paul Byerly	Instructor in Pediatrics
G. Bowers Mansdorfer	Instructor in Pediatrics
Jerome Fineman	Instructor in Pediatrics
Thomas A. Christensen	Instructor in Pediatrics
B. Matthew Debuskey	Instructor in Pediatrics
W. J. Schmitz	Assistant in Pediatrics
Israel P. Meranski	Assistant in Pediatrics
H. Whitney Wheaton	Assistant in Pediatrics
Lauriston L. Keown	Assistant in Pediatrics
Abraham Hurwitz	Assistant in Pediatrics
Israel Rosen	Assistant in Pediatrics
†Gibson J. Wells	Assistant in Pediatrics
‡Mabel G. Wilkin	Clinical Fellow in Pediatrics

Third Year. The course is presented as follows:

A. Lectures on infant feeding and the fundamentals of diseases of infants and children. (15 hours.)

B. Lectures on contagious diseases in conjunction with the Department of Hygiene and Preventive Medicine. (14 hours.)

C. A special course in physical diagnosis is given at City Hospitals. (20 hours.)

D. Clinical conferences demonstrating diseases of the newly-born. (6 hours.)

Fourth Year. A. An Amphitheatre Clinic is given at which patients are shown to demonstrate the features of the diseases discussed. (30 hours.)

B. Conferences and demonstrations are given in problems concerning diagnosis, care, treatment and clinical pathology of the diseases of infants and children. (30 hours.)

Students are assigned subjects for the preparation of theses.

C. Clinical clerkships are assigned on the pediatric wards, where experience is gained in taking histories, making physical examinations, doing routine laboratory work, and following up the patients' progress. This is under the supervision of the visiting staff. (140 hours.)

D. Instruction is given in the pediatric clinic of the out-patient department of the University Hospital. This consists of 1½ hours daily for five weeks—30 minutes each day being devoted to a clinical demonstration of some interesting case by a member of the staff; one hour daily to taking histories and making a physical examination under supervision of one of the staff instructors. (45 hours.)

Total hours: 300.

NEUROLOGY

Irving J. Spear	Professor of Neurology
Andrew C. Gillis	Professor of Neurology
G. M. Settle	Associate Professor of Neurology and Clinical Medicine
§Milford Levy	Associate Professor of Neurology
Leon Freedom	Associate Professor of Neurology
Benjamin Pushkin	Assistant Professor of Neurology
Philip F. Lerner	Instructor in Neurology
Bernhard Badt	Instructor in Neurology
William L. Fearing	Instructor in Neurology
Charles E. Balfour	Assistant in Neurology
†Edward F. Cotter	Assistant in Neurology
†Harry B. Teitelbaum	Assistant in Neurology
†John W. Machen	Assistant in Neurology

Second Year. Fifteen one-hour lectures are given to correlate the anatomy and physiology of the nervous system with clinical neurology.

Third Year. Twenty hours of instruction are given to the whole class in neuro-pathology supplemented with pathological demonstrations. Sixteen lecture-demonstrations are given in which the major types of the diseases of the nervous system are discussed. A course is also given at the Baltimore City Hospitals, consisting of six periods of two hours each, in which the students in small groups carry out complete neurological examinations of selected cases which illustrate the chief neurological syndromes.

Fourth Year. A clinical conference one hour each week is given to the whole class at the University and Mercy Hospitals. All patients presented at these clinics are carefully examined. Complete written records are made by the students who demonstrate the patients before the class. The patients are usually assigned one or two weeks before they are presented, and each student in the class must study and present one or more patients during the year.

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§ Deceased, October 10, 1939.

Ward Class Instruction. Nine hours of instruction are given to each student in small sections at the University and Mercy Hospitals. In these classes the students come in close personal contact with the patients in the wards under the supervision of the instructor.

Dispensary Instruction. Small sections are instructed in the dispensaries of the University and Mercy Hospitals five afternoons each week. In this way students are brought into contact with nervous diseases in their early and late manifestations.

HYGIENE AND PUBLIC HEALTH

Huntington Williams	Professor of Hygiene and Public Health
William H. F. Warthen	Assistant Professor of Hygiene and Public Health
Myron G. Tull	Instructor in Hygiene and Public Health

Third Year. A one-hour lecture is given to the whole class each Monday from September to May. Basic instruction is given in the clinical and public health aspects of the communicable diseases. The lectures are under the auspices of the Department of Medicine and are given by staff members of that department, including physicians representing Pediatrics and Hygiene and Public Health.

Fourth Year. Two-hour instruction periods are devoted to the whole class, in groups of ten to fifteen students, on six Wednesday afternoons. These sessions enable the students themselves to prepare birth and death certificates, to vaccinate against smallpox and to conduct other practical public health procedures. In addition there are four Wednesday afternoon field inspection trips for each third of the class. These trips, under guidance of full time public health workers, include visits to (1) city water filtration plant, (2) sewage disposal plant, (3) milk pasteurization plant and bakery and (4) industrial plant which has an active program of hygiene. Elective case work is assigned the Western Health District.

The course deals with the fundamentals of public health and supplements the work in the third year. The major emphasis in both years is on the practice of preventive medicine and the relation of prevention to diagnosis and treatment. The whole class, in small groups, receives practical instruction at Sydenham Hospital, the one-hundred bed communicable disease hospital of the Baltimore City Health Department.

MEDICAL JURISPRUDENCE

Howard J. Maldeis	Associate Professor of Medical Jurisprudence Chief Medical Examiner, Maryland

Third Year. This course embraces a summary of some of the following: Proceedings in criminal and civil prosecution, medical evidence and testimony, identity and its general relations, personal identity, rape, criminal abortions, signs of death, wounds in their medico-legal relations, natural and homocidal death, malpractice, insanity, and medico-legal autopsies.

Total hours: 3.

DEPARTMENT OF SURGERY

Arthur M. Shipley	Professor of Surgery
Walter D. Wise	Professor of Surgery
Page Edmunds	Professor of Traumatic Surgery
Charles Bagley, Jr.	Professor of Neurological Surgery
Elliott H. Hutchins	Professor of Clinical Surgery
Charles Reid Edwards	Professor of Clinical Surgery
F. L. Jennings	Professor of Clinical Surgery
§Robert P. Bay	Professor of Oral Surgery
Thomas B. Aycock	Clinical Professor of Surgery
Thomas R. Chambers	Associate Professor of Surgery
R. W. Locher	Associate Professor of Clinical Surgery
Edward S. Johnson	Associate Professor of Surgery
D. J. Pessagno	Associate Professor of Surgery
Charles A. Reifschneider	Associate Professor of Traumatic Surgery
Monte Edwards	Associate Professor of Surgery
N. Clyde Marvel	Associate Professor of Surgery
Richard G. Coblentz	Associate Professor of Neurological Surgery
Grant E. Ward	Associate Professor of Surgery
Cyrus F. Horine	Associate Professor of Surgery
George H. Yeager	Assistant Professor of Surgery
H. F. Bongardt	Assistant Professor of Surgery
I. O. Ridgley	Associate in Surgery
C. W. Peake	Associate in Surgery
Henry F. Ullrich	Associate in Surgery
William R. Johnson	Instructor in Surgery
E. M. Hanrahan	Instructor in Surgery
S. Demarco	Instructor in Surgery
Karl J. Steinmueller	Instructor in Surgery
Luther E. Little	Instructor in Surgery
J. Frank Hewitt	Instructor in Surgery
Simon H. Brager	Instructor in Surgery
James W. Nelson	Instructor in Surgery
I. Ridgeway Trimble	Instructor in Surgery
Richard T. Shackelford	Instructor in Surgery
Raymond F. Helfrich	Instructor in Surgery
Harry C. Hull	Instructor in Surgery
†Murray M. Copeland	Instructor in Surgery
†E. Eugene Covington	Instructor in Surgery

§ Deceased January 1, 1940.

†J. Duer Moores	Instructor in Surgery
William R. Geraghty	Assistant in Surgery
Howard B. McElwain	Assistant in Surgery
A. V. Buchness	Assistant in Surgery
T. J. Touhey	Assistant in Surgery
Clyde F. Karns	Assistant in Surgery
Samuel H. Culver	Assistant in Surgery
Albert R. Wilkerson	Assistant in Surgery
J. H. Wilkerson	Assistant in Surgery
L. T. Chance	Assistant in Surgery
Samuel McLanahan, Jr.	Assistant in Surgery
W. Allen Deckert, Jr.	Assistant in Surgery
Samuel E. Proctor	Assistant in Surgery
George Govatos	Assistant in Surgery
Dwight Currie	Assistant in Surgery
Robert F. Healy	Assistant in Surgery
Herbert M. Foster	Assistant in Surgery
Otto C. Brantigan	Assistant in Surgery
Daniel R. Robinson	Assistant in Surgery
Robert F. Chenowith	Assistant in Surgery
Joseph V. Jerardi	Assistant in Surgery
Thurston R. Adams	Assistant in Surgery
†Eldred Roberts	Assistant in Surgery
†Alexander McGill Duff	Assistant in Surgery
†Arthur G. Siwinski	Assistant in Surgery

Instruction is given by means of lectures, laboratory work, recitations, dispensary work, bedside instruction, ward classes, and clinics. The work begins in the second year, and continues throughout the third and fourth years.

The teaching is done in the anatomical laboratory, operative surgery laboratory, the dispensaries, wards, clinical laboratories and operating rooms of the University and Mercy Hospitals, and in the wards and operating rooms of the Baltimore City Hospitals.

SECOND YEAR

Topographic and Surgical Anatomy. The course is designed to bridge the gap between anatomy in the abstract and clinical anatomy as applied to the study and practice of medicine and surgery.

The teaching is done in the anatomical laboratory, and students are required to demonstrate all points, outlines, and regions on the cadaver. Underlying regions are dissected to bring out outlines and relations of structures.

Two lectures and two laboratory periods per week during the second semester. Drs. Monte Edwards, Hull, Brantigan and Adams.

Total hours: 96.

Principles of Surgery. This course includes history-taking, records of physical examinations, and of operations and progress notes; the preparation of surgical dressings, suture materials and solutions. It includes inflammation, infections, ulcers, gangrene, fistulae and sinuses, hemorrhage, shock and tumors. Lectures and conferences, two hours per week for one semester, are given to the whole class. Dr. Aycock.

THIRD YEAR

General and Regional Surgery. Lectures, recitations and clinics on the principles of surgery and general surgery are given three hours a week to the whole class. Dr. C. R. Edwards.

The class is divided into groups and receives instruction in history-taking, gross pathology, and surgical diagnosis—at the bedside and in the dead-house of the Baltimore City Hospitals. Drs. Aycock, Reifschneider, Toulson, Hull, Yeager and Brantigan.

Operative Surgery. Two courses are given in Operative Surgery under the supervision of Dr. Yeager assisted by Drs. Peake, Ullrich, Brager, Brouillet, Chenowith, Culver, Deckert, Foster, Govatos, Healy, Jerardi, Karns, Mech and Williamson. The class is divided into sections and each section is given practical and individual work under the supervision of the instructors.

Fractures and Dislocations. This course consists of instruction in the various forms of fractures, dislocations and their treatment. There is a regular schedule of didactic lectures, which is supplemented by practical demonstrations in diagnosis and treatment.

Surgical Dispensary. Under supervision, the student takes the history, makes the physical examinations, attempts the diagnosis, and, as far as possible, carries out the treatment of the ambulatory surgical patients in the University and in the Mercy Hospitals. Mercy Hospital—Dr. Helfrich assisted by the whole dispensary staff. University Hospital—Dr. Yeager assisted by the whole dispensary staff.

FOURTH YEAR

Clinics. A weekly clinic is given at the Mercy and at the University Hospitals to one-half the class throughout the year. As far as possible this is a diagnostic clinic. Mercy Hospital—Dr. Wise. University Hospital—Dr. Shipley.

Surgical Pathology. A weekly exercise of one hour at Mercy Hospital for one semester at which specimens from the operating room and museum are studied in the gross and microscopically in relation to the case history. Dr. Pessagno.

Traumatic Surgery. This course deals with operative and post-operative treatment of accident cases, with instructions as to the relationship between the state, the employee, the employer, and the physician's duty to each. One hour a week to sections of the class throughout the year. Dr. Edmunds.

Clinical Clerkship. This work includes the personal study of assigned hospital patients, under supervision of the staffs of the University and Mercy Hospitals, history-taking, and physical examination of patients, laboratory examinations, attendance at operations and observation of post-operative treatment.

Ward Classes. Ward-class instruction in small groups will consist of ward rounds, surgical diagnosis, treatment and the after-care of operative cases. Mercy Hospital—Drs. Wise, Hutchins, Jennings, Pessagno and Marvel. University Hospital—Drs. Shipley, Edmunds, Edwards and Aycock.

ANAESTHESIA

S. Griffith Davis	Professor of Anaesthesia
E. Hollister Davis	Assistant in Anaesthesia
Mary J. O'Brien	Anaesthetist
Ruth Elliott	Anaesthetist

THIRD YEAR

Lectures are given on the general physiology of anaesthesia, with consideration of special physiology of each anaesthetic agent; methods of induction and administration of anaesthesia; factors influencing the selection of the anaesthetic and types of anaesthetic agents; and the preparation and care of the anaesthetized patient.

The lectures are correlated with practical demonstrations during operative clinics at the City Hospitals.

FOURTH YEAR

During operative clinics in both surgery and gynecology each student will be given practical instruction in the administration of anaesthetics and will be required to record such changes as take place in blood pressure, pulse and respiration.

DERMATOLOGY

Harry M. Robinson	Professor of Dermatology
*Francis Ellis	Assistant Professor of Dermatology
John R. Abercrombie	Associate in Dermatology
Harold M. Goodman	Associate in Dermatology
*Joseph C. Bernstein	Associate in Dermatology
Harry M. Robinson, Jr.	Instructor in Dermatology
Rollin C. Hudson	Assistant in Dermatology
Thomas E. Roach	Assistant in Dermatology
Jeannette R. Heghinian	Assistant in Dermatology
†Lester N. Kolman	Assistant in Dermatology
Reuben Rochkind	Fellow in Dermatology

A weekly clinic is given at University and Mercy Hospitals throughout the year. This course consists of demonstrations of the common diseases of the skin in addition to a number of lectures on the general principles of dermatology. Drs. Robinson and Ellis.

Dispensary instruction is given in the diagnosis and treatment of skin diseases at the University Hospital by Dr. Robinson and dispensary staff; and at Mercy Hospital by Dr. Ellis and dispensary staff.

Third year	15 hours
Fourth Year	49 hours
Total	64 hours

ORTHOPAEDIC SURGERY

Allen Fiske Voshell	Professor of Orthopaedic Surgery
Albertus Cotton	Professor of Orthopaedic Surgery
Compton Riely	Clinical Professor of Orthopaedic Surgery
Moses Gellman	Associate Professor of Orthopaedic Surgery
Harry L. Rogers	Associate Professor of Orthopaedic Surgery
H. Alvan Jones	Associate in Orthopaedic Surgery
Henry F. Ullrich	Associate in Orthopaedic Surgery
†John V. Hopkins	Assistant in Orthopaedic Surgery

Didactic, clinical, bedside and out-patient instruction is given to the Fourth Year at the University and Mercy Hospitals and Dispensaries, Kernan Hospital for Crippled Children at Dickeyville and Baltimore City Hospitals. Instruction is also given to the Third Year in small groups at the Baltimore City Hospitals.

Weekly lectures throughout the year present all phases of orthopaedic surgery except fractures; brief discussions and demonstrations of physical therapy are included.

Fourth Year groups are given more intimate instruction biweekly at one of the above institutions; fracture cases are included here.

Third year	60 hours
Fourth year	90 hours
Total	150 hours

ROENTGENOLOGY

Henry J. Walton	Professor of Roentgenology
Albertus Cotton	Professor of Roentgenology
Walter L. Kilby	Assistant Professor of Roentgenology
Eugene L. Flippin	Associate in Roentgenology

During the academic year small groups of the fourth year class are given weekly instruction in the diagnostic and therapeutic uses of the Roentgen rays. An effort is made to familiarize the student with the indications for and limitations of Roentgen ray examinations. The history, physics and practical application of Roentgen rays are alluded to but not stressed. Conferences are held with the various departments during the school year which are also open to members of the fourth year class.

Total hours: 96.

DISEASES OF THE NOSE AND THROAT

Edward A. Looper	Professor of Diseases of the Nose and Throat
Waitman F. Zinn	Clinical Professor of Diseases of the Nose and Throat
Franklin B. Anderson	Associate Professor of Diseases of the Nose and Throat
W. Raymond McKenzie	Associate in Diseases of the Nose and Throat
Thomas R. O'Rourk	Associate in Diseases of the Nose and Throat
Cleo D. Stiles	Assistant in Diseases of the Nose and Throat

Third Year. Instruction to whole class is given in the common diseases of the nose and throat, attention being especially directed to infections of the accessory sinuses, the importance of focal infections in the etiology of general diseases and modern methods of diagnosis. Lectures illustrated by lantern slides are given one hour weekly for seven weeks by Dr. Looper.

Fourth Year. Dispensary instruction is given for one and one-half hours daily, to small sections at the University and the Mercy Hospitals. The student is afforded an opportunity to study, diagnose and treat patients under supervision. Ward classes and clinical demonstrations are given in periods of one and one-half hours weekly throughout the session in the University and the Mercy Hospitals.

The Looper Clinic for bronchoscopy and esophagoscopy, recently established in the University Hospital, affords unusual opportunities for students to study diseases of the larynx, bronchi and esophagus. The clinic is open to students daily from 2 to 4 P.M., under direction of Dr. Looper.

The Mercy Hospital Clinic for bronchoscopy and esophagoscopy is under the direction of Dr. Zinn. In these two clinics the etiology, symptomatology, diagnosis and treatment of foreign bodies in the air and food passages, as well as bronchoscopy, are taught to students, as an aid in the diagnosis and treatment of diseases of the lungs.

Third year	9 hours
Fourth year	53 hours
Total	62 hours

GENITO-URINARY SURGERY

W. H. Toulson	Professor of Genito-Urinary Surgery
Kenneth D. Legge	Associate Professor of Genito-Urinary Surgery
Austin H. Wood	Associate in Genito-Urinary Surgery
L. J. Millan	Associate in Genito-Urinary Surgery
L. K. Fargo	Associate in Genito-Urinary Surgery

John F. Hogan	Associate in Genito-Urinary Surgery
W. A. H. Councill	Associate in Genito-Urinary Surgery
Samuel T. Helms	Instructor in Genito-Urinary Surgery
Harry S. Shelley	Instructor in Genito-Urinary Surgery
Francis W. Gillis	Instructor in Genito-Urinary Surgery
†Hugh Jewett	Assistant in Genito-Urinary Surgery
†Howard B. Mays	Assistant in Genito-Urinary Surgery

Third Year. This course is given for seven hours to the whole class. It consists of lectures and demonstrations, including the use of lantern slides and motion pictures. Dr. Toulson.

Fourth Year. The course in this year includes explanations and demonstrations of urethroscopy, cystoscopy, ureteral catheterization, renal function tests, urography, urine cultures and the various laboratory procedures. The teaching consists of clinics and ward rounds to small groups, and attendance by members of the senior class upon the out-patients in the dispensary. The student here is placed much on his own responsibility in arriving at a diagnosis. Members of the Staff are in constant attendance for consultations. These dispensary classes are conducted at both the Mercy and University Hospitals where practically every variety of urogenital disease is encountered and used for teaching purposes.

Third year	8 hours
Fourth year	64 hours
Total	72 hours

DISEASES OF THE RECTUM AND COLON

Charles F. Blake	Professor of Diseases of Rectum and Colon
J. Dawson Reeder	Professor of Diseases of Rectum and Colon
Monte Edwards	Associate Professor of Diseases of Rectum and Colon
James C. Owings	Instructor in Diseases of the Rectum and Colon

Third Year. Seven lectures are given to the whole class. This course is for instruction in the diseases of the colon, sigmoid flexure, rectum and anus, and covers the essential features of the anatomy and physiology of the large intestine as well as the various diseases to which it is subject. Dr. Reeder and Dr. Edwards.

Fourth Year. Ward and dispensary instruction is given in the University and Mercy Hospitals, where different phases of the various diseases are taught by direct observation and examination. The use of the proctoscope and sigmoidoscope in the examination of the rectum and sigmoid is made familiar to each student. Mercy Hospital—Dr. Blake. University Hospital—Drs. Reeder and Monte Edwards.

Third year	7 hours
Fourth year	16 hours
Total	23 hours

OTOLOGY

J. W. Downey	Professor of Otology
Franklin B. Anderson	Associate Professor of Otology
Benjamin S. Rich	Associate in Otology
Thomas R. O'Rourk	Associate in Otology
William A. Parr	Assistant in Otology
†Cleo D. Stiles	Assistant in Otology

The course in otology is planned to give a practical knowledge of the anatomy and physiology of the ear, and its proximity and relationship to the brain and other vital structures. The inflammatory diseases, their etiology, diagnosis, treatment and complications are particularly stressed, with emphasis upon their relationship to the diseases of children, head-surgery and neurology.

Third Year. The whole class is given instruction by means of talks, anatomical specimens and lantern slides.

Fourth Year. Small sections of the class receive instruction and make personal examinations of patients under the direction of an instructor. The student is urged to make a routine examination of the ear in his ward work in general medicine and surgery.

Third year	12 hours
Fourth year	40 hours
Total	52 hours

NEUROLOGICAL SURGERY

Charles Bagley, Jr.	Professor of Neurological Surgery
Richard G. Coblentz	Associate Professor of Neurological Surgery
†Edward F. Cotter	Hitchcock Fellow in Neuro-surgery

Third Year. The course covers instruction in diagnosis and treatment of surgical conditions of the brain, spinal cord and the peripheral nerves. Dr. Bagley.

Fourth Year. Weekly ward rounds and conferences are given at the University Hospital. Drs. Bagley and Coblentz.

Third year	12 hours
Fourth year	30 hours
Total	42 hours

ONCOLOGY

J. Mason Hundley, Jr.	Professor of Gynecology
Grant E. Ward	Associate Professor of Surgery
†Murray M. Copeland	Instructor in Surgery
†E. Eugene Covington	Instructor in Surgery
†J. Duer Moores	Instructor in Surgery
James W. Nelson	Instructor in Surgery

*Beverley C. Compton	Instructor in Gynecology
*John C. Dumler	Instructor in Gynecology
†Eldred Roberts	Assistant in Surgery
Alexander McGill Duff	Fellow of the National Cancer Institute
Arthur G. Siwinski	Fellow of the National Cancer Institute in absentia

Every facility for the diagnosis and treatment of neoplastic diseases is available; this includes electro-surgery, radium therapy and deep X-ray therapy.

An out-patient clinic is held twice weekly which affords an opportunity for instruction to a limited number of students. The gynecological problems are under the supervision of Dr. Hundley, and the general surgical conditions are under the direction of Dr. Ward.

Instruction, other than dispensary teaching, is given to small groups of students, for one hour a week, in the history, physics and practical application of radium. Drs. Ward and Hundley.

Third year	8 hours
Fourth year	16 hours
Total	24 hours

[1]ORAL SURGERY

§Robert P. Bay	Professor of Oral Surgery
Brice M. Dorsey	Professor of Exodontia
Charles A. Reifschneider	Associate Professor of Traumatic Surgery and Oral Surgery
William E. Hahn	Instructor in Exodontia

This section in the Department of Surgery is established for the teaching of both medical and dental students. A new subdivision in the Dispensary has also been established. Beds will be provided in the University Hospital for the care of patients who will be available for the teaching of students from both schools.

Senior year: clinics weekly.

Ward instruction and group teaching are given. This includes diagnosis and treatment of diseases of the face, mouth and jaws.

INDUSTRIAL MEDICINE AND SURGERY

Page Edmunds	Professor of Traumatic Surgery
G. Carroll Lockard	Professor of Clinical Medicine
Charles A. Reifschneider	Associate Professor of Traumatic Surgery and Oral Surgery
Thurston R. Adams	Assistant in Surgery

This section is under the combined supervision of the Medical and Surgical Departments. It is a cooperative effort by members of the Medical

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[1] Discontinued 1940–1941.

§ Deceased January 1, 1940.

School and Hospital Staff to afford means for clinical and laboratory study of the patient who has been subjected to traumatic or medical industrial hazard, so that adequate care may be instituted to promote his physical well-being. The facilities of the Laboratories of the Medical School and Hospital are available as required.

Under direction of this department limited undergraduate instruction is given, especially in the methods of examination and of keeping records; and in the general medico-legal principles as they affect the industrial employee, the employer, the general insurers, the physician and the hospital. There is also instruction on methods of making life insurance and other physical examinations, whether for employment or for health purposes. The wards of the University, Mercy and City Hospitals provide for bed-side instruction.

Total hours: 48.

DEPARTMENT OF OBSTETRICS

L. H. Douglass	Professor of Obstetrics
J. McFarland Bergland	Associate Professor of Obstetrics
Emil Novak	Associate Professor of Obstetrics
J. G. M. Reese	Assistant Professor of Obstetrics
M. Alexander Novey	Assistant Professor of Obstetrics
Isadore A. Siegel	Assistant Professor of Obstetrics
E. P. H. Harrison	Associate in Obstetrics
John E. Savage	Associate in Obstetrics
Frank K. Morris	Instructor in Obstetrics
Margaret B. Ballard	Assistant in Obstetrics
Kenneth B. Boyd	Assistant in Obstetrics
Dudley P. Bowe	Assistant in Obstetrics
W. Allen Deckert	Assistant in Obstetrics
Jaroslav Hulla	Assistant in Obstetrics
Marius P. Johnson	Assistant in Obstetrics
Maxwell L. Mazer	Assistant in Obstetrics
Joseph M. Blumberg	Assistant in Obstetrics
Hugh B. McNally	Assistant in Obstetrics
Ferd. E. Kadan	Assistant in Obstetrics
J. Edward Norris	Assistant in Obstetrics
Jacob R. Jensen	Assistant in Obstetrics
J. Warren Albrittain	Assistant in Obstetrics

Third Year. The lectures and recitations consisting of three hours teaching weekly are designed to cover the anatomy of the female generative tract and the bony pelvis, the physiology and development of the ovum, and the physiology of pregnancy and labor. Following this the pathology of pregnancy, labor and the puerperium are taken up. Drs. Douglass, Novak, Reese, Novey, Savage and Siegel.

Each student spends time during his junior year at the Baltimore

City Hospitals observing, assisting and finally delivering patients under strict supervision. Each student sees about twenty deliveries there, and does a considerable amount of the routine work.

The junior students are assigned as assistants to the seniors in the home delivery service and accompany them on deliveries.

Each student receives, in small groups, ten hours instruction in palpation of patients and mensuration of the pelvis and demonstrations of the mechanism of labor. Drs. Siegel and Harrison.

Fourth Year. At the weekly clinical conference, cases are presented and discussed and the student body is encouraged to offer opinions and to ask questions. There is no didactic teaching done, and an earnest effort is made to keep it, in every sense of the word, a conference. Dr. Douglass.

The ward classes are held three times weekly for five weeks for each group. Various subjects are assigned and discussed, patients and their histories are presented. Drs. Douglass, Reese and Novey.

Manikin instruction is given once a week. Drs. Morris, Bowe and Albrittain.

During the same five-week period, the students are sent into patients' homes to conduct deliveries under supervision of a senior member of the house staff and with the assistance of a graduate nurse. The student is held responsible for the complete conduct of each assigned case.

Each student spends thirty hours in the prenatal clinic, taking histories and examining patients under supervision.

Finally, the students are invited to attend the monthly meetings of The Committee on Maternal Mortality, where all maternal deaths occurring in Baltimore are openly discussed.

Third year	148 hours
Fourth year	102 hours
Total	250 hours

DEPARTMENT OF GYNECOLOGY

J. Mason Hundley, Jr.	Professor of Gynecology
Abram S. Samuels	Clinical Professor of Gynecology
Thomas K. Galvin	Assistant Professor of Gynecology
Leo Brady	Assistant Professor of Gynecology
R. G. Willse	Assistant Professor of Gynecology
George A. Strauss, Jr.	Assistant Professor of Gynecology
John T. Hibbitts	Associate in Gynecology
Edward P. Smith	Associate in Gynecology
Kenneth B. Boyd	Associate in Gynecology
Thomas S. Bowyer	Instructor in Gynecology
Joseph V. Castagna	Instructor in Gynecology
Ernest S. Edlow	Instructor in Gynecology

*John C. Dumler	Instructor in Gynecology
*Beverley C. Compton	Instructor in Gynecology
*W. Allen Deckert	Instructor in Gynecology
*Frank K. Morris	Instructor in Gynecology
*Houston Everett	Instructor in Gynecology
H. L. Granoff	Assistant in Gynecology
J. J. Erwin	Assistant in Gynecology

Third Year. A course of thirty lectures and recitations is given to the whole class. In addition, a short course of lecture-demonstrations is given at the Baltimore City Hospitals, consisting of eight periods of one hour each, in which small groups of students are instructed in the fundamentals of gynecological diagnosis and examination.

Fourth Year. Operative clinics—lectures and demonstrations—are given six hours per week, for five weeks, to sections of the class.

Instruction in Female Urology is given. A small number of students may attend the cystoscopic dispensary which is held twice weekly.

The course in Gynecology also includes instruction in the diagnosis and treatment of cancer of the generative organs. Small groups of students attend the oncological dispensary for additional work.

Third year	38 hours
Fourth year	74 hours
Total	112 hours

DEPARTMENT OF OPHTHALMOLOGY

Clyde A. Clapp	Professor of Ophthalmology
M. Randolph Kahn	Clinical Professor of Ophthalmology
H. K. Fleck	Clinical Professor of Ophthalmology
Jonas Friedenwald	Lecturer in Ophthalmic Pathology
R. D. West	Associate in Ophthalmology
Henry F. Graff	Associate in Ophthalmology
Joseph I. Kemler	Associate in Ophthalmology
F. A. Holden	Instructor in Ophthalmology
F. Edwin Knowles, Jr.	Instructor in Ophthalmology
John G. Runkle	Assistant in Ophthalmology
Thomas R. O'Rourk	Assistant in Ophthalmology
Jerome Snyder	Assistant in Ophthalmology
Milton C. Lang	Assistant in Ophthalmology
†Arnold L. Peter	Assistant in Ophthalmology

Third Year. Second semester. Dr. Kahn reviews the anatomy and physiology of the eye and discusses the methods used in making the various examinations. Errors of refraction and their effect upon the general system are explained. Weekly section work, demonstrating the use of the ophthalmoscope, is carried on during the entire session.

Fourth Year. Clinics and demonstrations are given in diseases of the eye, weekly, for one year. Dr. Clapp.

This course consists of lectures upon the diseases of the eye, with particular reference to their diagnosis and relation to general medicine. Special lectures will be given upon vascular changes in the eye and upon the pathology of the eye. Some operations will be demonstrated by motion pictures.

Weekly ward classes are held at the University, The Baltimore Eye, Ear and Throat and Mercy Hospitals during which the eye grounds in the various medical and surgical conditions are demonstrated. Also daily demonstrations are given in the taking of histories and the diagnosis and treatment of the various conditions as seen in the dispensary. Drs. Fleck, West, Kemler, Graff and Knowles.

Third year	20 hours
Fourth year	104 hours
Total	124 hours

ART AS APPLIED TO MEDICINE

Carl Dame Clarke, M. A. .... Associate Professor of Art as Applied to Medicine

This department is maintained for the purpose of supplying pictorial and plastic illustrations for visual teaching in the classrooms of the University and for publication in scientific periodicals.

Special courses of instruction are given to qualified students.

FIRST YEAR SCHEDULE

FIRST SEMESTER, SEPTEMBER 26, 1940 TO FEBRUARY 1, 1941

<table>
<tr><th>Hours</th><th>Monday</th><th>Tuesday</th><th>Wednesday</th><th>Thursday</th><th>Friday</th><th>Saturday</th></tr>
<tr><td>9.00 to 1.00</td><td colspan="5">Gross Anatomy—Lectures and Laboratories
Bressler 1</td><td>(9–12)</td></tr>
<tr><td>1.00 to 2.00</td><td colspan="5">Lunch</td><td></td></tr>
<tr><td>2.00 to 5.00</td><td colspan="2">*Histology and Embryology
Bressler 2</td><td></td><td colspan="2">Histology and Embryology
Bressler 2</td><td></td></tr>
</table>

* Course begins October 10, 1940.

SECOND SEMESTER, FEBRUARY 3 TO MAY 31, 1941

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 12.00	Laboratory — Biological Chemistry *Sect. A*	Laboratory — Biological Chemistry *Sect. B*	Laboratory — Biological Chemistry *Sect. A*	Laboratory — Biological Chemistry *Sect. B*	10.30 to 11.30 Psychiatry *Adm. 1*	
12.00 to 12.50	Lunch	Lunch	Lunch	Lunch	Lunch	
12.50 to 1.50	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	Biological Chemistry *Adm. 1*	
2.00 to 5.00	*Neuro-anatomy *Bressler 2*	Neuro-anatomy *Bressler 2*	Biological Chemistry Conference *Adm. 1*	Neuro-anatomy *Bressler 2*	Biological Chemistry Conference *Adm. 1*	

* Course begins March 3, 1941.

Locations of Lecture Halls and Laboratories:

Adm. 1—First Floor, Administration Building, Lombard and Greene Streets.
A. H.—Anatomical Hall—Upper Hall, N. E. Cor. Lombard and Greene Streets.
C. H.—Chemical Hall, Lower Hall, N. E. Cor. Lombard and Greene Streets.
Biological Chemistry Laboratory—Third Floor, 31 South Greene Street.
Bressler Research Laboratory—29 S. Greene Street.
  Gross Anatomy—First Floor
  Histology and Embryology—Second Floor.
  Neuro-anatomy—Second Floor.

Mid-Year Examinations—January 27-February 1, 1941

Final Examinations—May 26-31, 1941

SECOND YEAR SCHEDULE

FIRST SEMESTER, SEPTEMBER 26, 1940 TO FEBRUARY 1, 1941

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 9.30	Physiology *Bressler 2*	Physiology *Bressler 2*	Medicine *Bressler 2*	Physiology *Bressler 2*	Physiology *Bressler 2*	
9.30 to 10.30	Physiology Conference *Bressler 2*	Bacteriology *Adm. 1*	Physiology Conference *Bressler 2*	Pharmacology *Bressler 2*	Pharmacology *Bressler 2*	
10.30 to 11.30	†Bacteriology Laboratory				Psychiatry *C. H.* Neurological Diagnosis *C. H.*	
12.30	Lunch					
1.00 to 5.00	Physiology *A*	Laboratory *B*	Elective	Pharmacology (1 to 4) *B* Physiology *A*	Laboratory *A* Laboratory *B*	

† Bacteriology Laboratory—Section work during the last month.

SECOND SEMESTER, FEBRUARY 3 TO MAY 31, 1941

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	Surgery *Bressler 2*	Surgery *Bressler 2*	Surgical Anatomy *Bressler 2*			
9.30 to 10.30	Pharmacology *Bressler 2*	Pharmacology *Bressler 2*	Surgical Anatomy Laboratory *Bressler 1*	Medical Clinic *Amp.*	Pharmacology *Bressler 2*	
10.30 to 11.30	Pathology *C. H.*	Pathology *C. H.*		Pathology *C. H.*	Pathology *C. H.*	
11.30	LUNCH					
12.00 to 2.00	Pathology Laboratory	Pathology Laboratory	Immunology Laboratory	Pathology Laboratory	Pathology Laboratory	
2.00 to 3.00	Surgical Anatomy *Bressler 2*	‖Immunology Laboratory		Pharmacology Laboratory *Sect. A*	Pharmacology Laboratory *Sect. B*	
3.00 to 5.00	Surgical Anatomy Laboratory *Bressler 1*		Optional period Pathology Immunology	Physical Diagnosis *Sect. B* (3.00–5.00) *U. H. D.*	Physical Diagnosis *Sect. A* (3.00–5.00) *U. H. D.*	

‖ Immunology Laboratory—Section work during last two months.

Locations of Lecture Halls and Laboratories:

Adm. 1—First floor, Administration Building, Lombard and Greene Streets.
C. H.—Chemical Hall, Lower Hall, Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre, New University Hospital, Greene and Redwood Streets, Eighth Floor
Laboratories:
Physiology, Pharmacology, Surgical, Anatomy—Bressler Building.
Bacteriology, Immunology, Pathology, Second Floor, 31 S. Greene Street.
U. H. D.—University Hospital Dispensary, Old Hospital Building.

Mid-Year Examinations—January 27–February 1, 1941
Final Examinations—May 26–31, 1941

THIRD YEAR SCHEDULE

SEPTEMBER 26, 1940 TO MAY 31, 1941

SCHEDULE 1

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	(Whole Class) Obstetrics *C. H.* †Gynecology Apr. 21 to May 12	(Whole Class) Surgery *C. H.*	(Whole Class) Obstetrics *C. H.* †Gynecology Apr. 24 to May 15	(Whole Class) Surgery *C. H.*	(Whole Class) Pathology *C. H.*	(Whole Class) Surgery *C. H.* †Gynecology Feb. 8 to Mar. 29 inc.
9.30 to 10.00	Transfer to Baltimore City Hospitals					
10.00 to 12.00	Physical Diagnosis, Pathology, Neurology and Pediatrics at B. C. H.					
12.00 to 1.00	Transfer and Lunch	Transfer and Lunch	Lunch	Transfer and Lunch	Lunch	
1.00 to 2.00	(Whole Class) Otology, Proctology, Urology Nose & Throat, *C. H.*	(Whole Class) *Gynecology †Eye—10 wks. (Feb. 4 to April 8) †Oncology —5 wks. (April 22 to May 13) *C. H.*	Medical Clinic *B. C. H.*	(Whole Class) Clinical Pathology *Bressler 2*	Obstetrical Clinic *B. C. H.*	
2.00 to 4.00	(Whole Class) Pathology Laboratory *31*		Surgery (2.00 to 4.00) — Pediatrics (2.00 to 4.30) — Gynecology Orthopaedics Psychiatry (Subgroups of Surgery Group) (4.00 to 5.00)	(Whole Class) Clinical Pathology — Laboratory *Bressler 5*	Surgery (2.00 to 4.00) — Pediatrics (2.00 to 4.30) — Gynecology Orthopaedics Psychiatry (Subgroups of Surgery Group) (4.00 to 5.00)	
4.00 to 5.00	(Whole Class) Hygiene and Public Health *C. H.*	(Whole Class) Physical Diagnosis, Psychiatry, Legal Medicine *C. H.*				

* First Semester. † Second Semester.

THIRD YEAR SCHEDULE
SEPTEMBER 26, 1940 TO MAY 31, 1941

SCHEDULE 2

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
8.30 to 9.30	Same as Schedule 1					
9.30 to 10.20	Pediatrics *C. H.*	Medicine *C. H.*	Medicine *C. H.*	Therapeutics *C. H.*	Medicine *C. H.*	Neurology *C. H.*
10.30 to 12.30	Operative Surgery—*Bressler 6* Medical and Surgical Dispensaries—(Univ. and Mercy Sections)					
12.30 to 1.00	LUNCH					
1.00 to 2.00	Same as Schedule 1		Medical Clinic *Amp.*	Same as Schedule 1	Psychiatry (9 weeks) Dermatology (6 weeks) N.B.—The whole section reports to psychiatry for first three weeks, then subdivides. *C. H.* for first 3 wks. U. H. Disp. Last 12 wks.	
2.00 to 4.00			Ophthalmoscopy (5 weeks) *B. E. H.* — Obstetrics (5 weeks) Univ. Hosp. Disp. — Otology (5 wks.) *A. H.*			
4.00 to 5.00					Obstetrics *C. H.*	

The Junior Class will be divided into two sections—A and B. Each section reports to classes in keeping with the following schedule assignment, in which the letters represent the class sections and the numerals indicate the schedules to be followed for the 15-week periods shown.

Schedule Assignment

Periods	*Sections and Schedules*
September 26 to January 25	A-1, B-2
February 3 to May 17	B-1, A-2

Locations of Lecture Halls, etc.

A. H.—Anatomical Hall, Upper Hall, N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre, New University Hospital, Eighth Floor.
B. C. H.—Baltimore City Hosps., 4940 Eastern Ave.
B. E. H.—Baltimore Eye, Ear and Throat Hospital, 1214 Eutaw Place.
Bressler—Bressler Building, 29 S. Greene Street.
C. H.—Chemical Hall, Lower Hall, N. E. Cor. Lombard and Greene Streets.
Univ. Hosp.—New University Hospital, Greene and Redwood Streets.
U. H. Disp.—Old Hospital Building, S. W. Cor. Lombard and Greene Streets.
31—31 South Greene Street.
Clinical Pathology Laboratory—Fifth Floor, Bressler Building.
Pathology Laboratory—31 South Greene Street, Special Rooms, Basement.

Mid-Year Examinations—January 27 to February 1, 1941
Final Examinations—May 19 to 31, 1941

FOURTH YEAR SCHEDULE

SEPTEMBER 26, 1940 TO MAY 31, 1941

Hours	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday
9.00 to 11.00	Ward Classes — (a) Medicine (a) Surgery (b) Obstetrics (d) Pediatrics	Ward Classes — (a) Medicine (a) Surgery (b) Gynecology (c) Gynecology	Ward Classes — (a) Medicine (a) Surgery (b) Obstetrics (d) Pediatrics	Ward Classes — (a) Medicine (a) Surgery (b) Gynecology (c) Gynecology	Ward Classes — (a) Medicine (a) Surgery (b) Obstetrics (d) Pediatrics	Ward Classes — (a) Medicine (a) Surgery (b) Gynecology (d) Pediatrics
11.00 to 12.00	Orthopaedic Surgery *Univ. Sec. Amp.* *P. & S. Sec. 51*	Medical Clinic *Univ. Sec. Amp.* Surgical Pathology *P. & S. Sec. 40*	Clinical Pathological Conference *Univ. Sec. C. H.* *P. & S. Sec. 34*	Surgical Clinic *Univ. Sec. Amp.* *P. & S. Sec. 51*	Medical Clinic *Univ. Sec. Amp.* *P. & S. Sec. 34*	Pediatric Clinic *Univ. Sec. Amp.* *P. & S. Sec. 34*
12.00 to 2.00	Dispensary Lunch and Transfer	Dispensary and Lunch	Dispensary Lunch and Transfer	Dispensary and Lunch	Dispensary Lunch and Transfer	Dispensary
2.15 to 3.15	Dermatology Clinic *Univ. Sec.* { *Disp. Amp.* *P. & S. Sec. 34*	Neurology Clinic *Univ. Sec. Amp.* *P. & S. Sec. 34*	Eye and Ear Clinic (Full Class at Univ. Hosp.) *Amp.*	Obstetrical Clinic (Full Class at Univ. Hosp.) *Amp.*	Hygiene and Public Health Sept. 22 to Oct. 27 Gastro-Enterology Clinic Nov. 3 (Full Class at Univ. Hosp.) *Amp.*	
3.30 to 5.00	*P. & S. Sect.* and *Univ. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	*P. & S. Sect.* Ward Classes	
		Medicine	Public Health	Medicine	Neurology	
		Orthopaedics	Nose and Throat	Proctology	Roentgenology	
		Pediatrics		Pediatrics	Psychiatry *Amp.*	
3.30 to 5.00	See special schedule Medical School bulletin board	*Univ. Sect.* Ward Classes	*Univ. Sect.* Ward Classes	*Univ. Sect.* Ward Classes	*Univ. Sect.* Ward Classes	
		Medicine	Hygiene and Public Health	Therapeutics	Neurology	
		Proctology	Urology	Nose and Throat	Orthopaedic Surgery (*Kernan Hospital*)	
		Oncology (3.30–4.30) *Amp.*	Eye and Ear		Psychiatry	

(a)—Univ. and P. & S. Sections.
(b)—Univ. Section.
(c)—Whole P. & S. special group.
(d)—P. & S. special group, divided attendance at Univ. and P. & S.

The Senior Class is divided into two sections, which report, one at Lombard and Greene Streets, the other at Calvert and Saratoga Streets, for one semester each, then rotate.

Each section of the class is divided into three groups—Medical, Surgical, and Special. These groups will rotate on the following dates:

First Semester		*Second Semester*	
1st period	Sept. 26–Nov. 2	1st period	Feb. 3–Mar. 8
2nd period	Nov. 4–Dec. 7	2nd period	Mar. 10–Apr. 9
3rd period	Dec. 9–Jan. 25	3rd period	Apr. 16–May 17

C. H.—Chemical Hall—N. E. Cor. Lombard and Greene Streets.
Amp.—Wilson Memorial Amphitheatre—New University Hospital.
Disp. Amp.—Old Hospital Building, fourth floor S. W. cor. Lombard and Greene Streets.
P. & S., 34—Second Floor, Calvert and Saratoga Streets.
P. & S., 40, 51—Fourth Floor, Calvert and Saratoga Streets.

Univ. Section-Dispensary schedule posted in Old Hospital Bldg.
P. & S. Section—obtain special schedule at Mercy Hospital

Mid-Year Examinations—January 27–February 1, 1941
Final Examinations—May 19–24, 1941

GRADUATES, UNIVERSITY OF MARYLAND SCHOOL OF MEDICINE AND COLLEGE OF PHYSICIANS AND SURGEONS, JUNE 3, 1939

Baylus, Herman Herbert	Maryland
Beck, Harry McBrine, A.B.	Maryland
Berman, Edgar Frank	Maryland
Bernstein, Aaron, B.S.	Maryland
Bernstein, Albion Older, B.S.	New York
Bess, Elizabeth Grant, A.B.	West Virginia
Bloom, Max Ralph, B.S.	Pennsylvania
Brezinski, Edward Joseph, A.B.	New Jersey
Briele, Henry Alison	Maryland
Brodsky, Bernard, A.B.	New York
Cannon, Lawrence Saville, A.B.	Utah
Cianos, James Nicholas, B.S.	Maryland
Coffman, Robert Thornhill	West Virginia
Cohen, Frank Samuel	Maryland
Corbitt, Richard Wylie, A.B.	West Virginia
Cunningham, Raymond Murray, A.B.	Maryland
Filtzer, David Leonard, A.B.	Maryland
Freed, Arnold Ulysses, B.S.	Maryland
Gaver, Leo Junior, B.S.	Maryland
Goldberg, Sylvan David, B.S.	Maryland
Grier, George Smith, III, B.S.	Delaware
Grott, Harold Allan, B.S.	Maryland
Haimowitz, Samuel Isaac, A.B.	Pennsylvania
Harris, Charles Isaac, Jr., B.S.	Georgia
Harrison, Charles Samuel, B.S.	West Virginia
Hartman, Oscar, B.S.	Maryland
Hartz, Alvin Sidney, A.B.	Maryland
Heimoff, Leonard Lincoln, A.B.	New York
Hooker, Charles Bullard, B.S.	Maryland
Hutchins, Thomas Manning	Maryland
Isaacson, Benjamin	Maryland
Jandorf, R. Donald, A.B.	Maryland
Jannarone, Lewis Henry, B.S.	New Jersey
Jones, Charles Wilson, A.B.	Maryland
Kairys, David, A.B.	Maryland
Kammer, William Henry, Jr., A.B.	Maryland
Kappelman, Melvin Daniel, B.S.	Maryland
Keister, Philip Weyforth, A.B.	Maryland
Kerr, James Patterson, Jr.	Maryland
Kiely, James Arthur, A.B.	New York
Kinnamon, Howard Franklin, Jr.	Maryland
Kleiman, Bernard Stanley	Maryland
Lapinsky, Herbert, M.S.	New York
Lavenstein, Arnold Fabian, A.B.	Maryland
Layman, William Templeton	Maryland
Leitch, William Harvey, B.S.	Maryland
Magness, Stephen Lee, A.B.	Maryland
Magruder, John Robinson, A.B.	Maryland
Marks, Irving Lowell	Maryland
McClafferty, William James, Jr., A.B.	Rhode Island
McLaughlin, Francis Joseph	Maryland
Meyer, Alvin Francis, B.S.	New York
Miller, Irving, B.S.	New York
Miller, William Shepherd, A.B.	Maryland
Moran, John Anthony	Massachusetts
Moricle, Charles Hunter, B.S.	North Carolina
Nuttall, James Baker, B.S.	Maryland
Palmer, David Waugh	West Virginia
Parks, Seigle Wilson, A.B.	West Virginia
Pijanowski, Walter Joseph, B.S.	New York
Pillar, Samuel, B.S.	Maryland
Polek, Melvin Frank, B.S.	Maryland
Reimann, Dexter LeRoy, B.S.	Maryland
Rochberg, Samuel, B.S.	New Jersey
Ruzicka, Edwin Russell, B.S.	Maryland
Sadove, Max Samuel, B.S.	Maryland
Schenthal, Joseph Edwin, A.B.	Maryland
Scher, Isadore	Maryland
Sexton, Thomas Scott, A.B.	West Virginia
Sherman, Claude Porter, B.S.	North Carolina
Siegel, Maurice Bert, A.B.	New York
Smoak, Philip Laurens	Florida
Solarz, Sylvan Daniel, A.B.	Maryland
Spiegel, Herbert, B.S.	Pennsylvania
Steger, William Joseph, A.B.	West Virginia
Stevens, Leland Bates, B.S.	Maryland
Tartikoff, George, B.S.	New York
Thomas, Ramsay Berry, B.S.	Maryland
Thomas, Wilbur Clyde, B.S.	Maryland
Urlock, John Peter, Jr., B.S.	Maryland
Wallenstein, Leonard, A.B.	Maryland
Wanner, Jesse Rosenberger, Jr.	Maryland
Whitworth, Fuller Barnard	Maryland
Wilder, Milton Jay	Maryland
Wilner, Sol, M.S.	New York
Worsley, Thomas Luther, Jr., B.S.	North Carolina
Zalis, Daniel Leonard, B.S.	Maryland

HONORS

University Prize Gold Medal.......................................JAMES BAKER NUTTALL

CERTIFICATES OF HONOR

FRANCIS JOSEPH MCLAUGHLIN
WILLIAM HARVEY LEITCH
LELAND BATES STEVENS
RAYMOND MURRAY CUNNINGHAM
THOMAS SCOTT SEXTON
RAMSAY BERRY THOMAS

The Dr. A. Bradley Gaither Memorial Prize of $25.00 for the best work in genito-urinary surgery during the senior year........................LEONARD WALLENSTEIN

INTERNSHIPS—GRADUATES OF 1939

Service begun July 1, 1939

Baylus, Herman Herbert	Columbia Hospital, Pittsburgh, Pennsylvania
Beck, Harry McBrine	Mercy Hospital, Baltimore, Maryland
Berman, Edgar Frank	Sinai Hospital, Baltimore, Maryland
Bernstein, Aaron	South Baltimore General Hospital, Baltimore, Maryland
Bernstein, Albion Older	Beth Israel Hospital, New York City
Bess, Elizabeth Grant	York Hospital, York, Pennsylvania
Bloom, Max Ralph	St. Francis Hospital, Pittsburgh, Pennsylvania
Brezinski, Edward Joseph	Newark City Hospital, Newark, New Jersey (begin 1/1/40), Perth Amboy General Hospital, Perth Amboy, New Jersey 7/1/39 to 12/31/39)
Briele, Henry Alison	University Hospital, Baltimore, Maryland
Brodsky, Bernard	Morrisania City Hospital, New York City
Cannon, Lawrence Saville	Maryland General Hospital, Baltimore, Maryland
Cianos, James Nicholas	St. Agnes Hospital, Baltimore, Maryland
Coffman, Robert Thornhill	Maryland General Hospital, Baltimore, Maryland
Cohen, Frank Samuel	Conemaugh Valley Memorial Hospital, Johnstown, Pennsylvania
Corbitt, Richard Wylie	Mercy Hospital, Baltimore, Maryland
Cunningham, Raymond Murray	University Hospital, Baltimore, Maryland
Filtzer, David Leonard	Baltimore City Hospital, Baltimore, Maryland
Freed, Arnold Ulysses	Sinai Hospital, Baltimore, Maryland
Gaver, Leo Junior	University Hospital, Baltimore, Maryland
Goldberg, Sylvan David	Baltimore City Hospital, Baltimore, Maryland
Grier, George Smith, III	Mercy Hospital, Baltimore, Maryland
Grott, Harold Allan	Sinai Hospital, Baltimore, Maryland
Haimowitz, Samuel Isaac	York Hospital, York, Pennsylvania
Harris, Charles Isaac	Emory University Hospital, Emory, Georgia
Harrison, Charles Samuel	Ohio Valley General Hospital, Wheeling, West Virginia
Hartman, Oscar	Franklin Square Hospital, Baltimore, Maryland
Hartz, Alvin Sidney	University Hospital, Baltimore, Maryland
Heimoff, Leonard Lincoln	Lincoln Hospital, Bronx, New York
Hooker, Charles Bullard	Emergency Hospital, Washington, D. C.
Hutchins, Thomas Manning	Maryland General Hospital, Baltimore, Maryland
Isaacson, Benjamin	Gallinger Municipal Hospital, Washington, D. C.
Jandorf, R. Donald	South Baltimore General Hospital, Baltimore, Maryland
Jannarone, Lewis Henry	Jersey City Medical Center, Jersey City, New Jersey
Jones, Charles Wilson	U. S. Marine Hospital, Baltimore, Maryland
Kairys, David	Beth Israel Hospital, New York City
Kammer, William Henry, Jr.	Mercy Hospital, Baltimore, Maryland

Kappelman, Melvin Daniel	Sinai Hospital, Baltimore, Maryland
Keister, Philip Weyforth	South Baltimore General Hospital, Baltimore, Maryland
Kerr, James Patterson, Jr.	Church Home & Infirmary, Baltimore, Maryland
Kiely, James Arthur	Binghamton City Hospital, Binghamton, New York
Kinnamon, Howard Franklin, Jr.	Mercy Hospital, Baltimore, Maryland
Kleiman, Bernard Stanley	St. Joseph's Hospital, Baltimore, Maryland
Lapinsky, Herbert	Lincoln Hospital, New York City
Lavenstein, Arnold Fabian	King's County Hospital, Brooklyn, New York
Layman, William Templeton	St. Mary's Hospital, Saginaw, Michigan
Leitch, William Harvey	University Hospital, Baltimore, Maryland
Magness, Stephen Lee	University Hospital, Baltimore, Maryland
Magruder, John Robinson	Baltimore City Hospital, Baltimore, Maryland
Marks, Irving Lowell	Lutheran Hospital, Cleveland, Ohio
McClafferty, William James, Jr.	Bon Secours Hospital, Baltimore, Maryland
McLaughlin, Francis Joseph	University Hospital, Baltimore, Maryland
Meyer, Alvin Francis	Coney Island Hospital, Brooklyn, New York
Miller, Irving	New York City Hospital, Welfare Island, New York
Miller, William Shepherd	University Hospital, Baltimore, Maryland
Moran, John Anthony	South Baltimore General Hospital, Baltimore, Maryland
Moricle, Charles Hunter	City Memorial Hospital, Winston-Salem, North Carolina
Nuttall, James Baker	University Hospital, Baltimore, Maryland
Palmer, David Waugh	St. Agnes Hospital, Baltimore, Maryland
Parks, Seigle Wilson	St. Agnes Hospital, Baltimore, Maryland
Pijanowski, Walter Joseph	Maryland General Hospital, Baltimore, Maryland
Pillar, Samuel	Sinai Hospital, Baltimore, Maryland
Polek, Melvin Frank	Mercy Hospital, Baltimore, Maryland
Reimann, Dexter LeRoy	Church Home & Infirmary, Baltimore, Maryland
Rochberg, Samuel	Passaic General Hospital, Passaic, New Jersey
Ruzicka, Edwin Russell	University Hospital, Baltimore, Maryland
Sadove, Max Samuel	St. Agnes Hospital, Baltimore, Maryland
Schenthal, Joseph Edwin	Mercy Hospital, Baltimore, Maryland
Scher, Isadore	West Baltimore General Hospital, Baltimore, Maryland
Sexton, Thomas Scott	Mercy Hospital, Baltimore, Maryland
Sherman, Claude Porter	Church Home & Infirmary, Baltimore, Maryland
Siegel, Maurice Bert	King's County Hospital, New York City
Smoak, Philip Laurens	Union Memorial Hospital, Baltimore, Maryland
Solarz, Sylvan Daniel	Mt. Sinai Hospital, Cleveland, Ohio
Spiegel, Herbert	St. Francis Hospital, Pittsburgh, Pennsylvania
Steger, William Joseph	Mercy Hospital, Pittsburgh, Pennsylvania
Stevens, Leland Bates	University Hospital, Baltimore, Maryland
Tartikoff, George	Israel Zion Hospital, Brooklyn, New York
Thomas, Ramsay Berry	University Hospital, Baltimore, Maryland
Thomas, Wilbur Clyde	Gallinger Municipal Hospital, Washington, D. C.
Urlock, John Peter, Jr.	South Baltimore General Hospital, Baltimore, Maryland
Wallenstein, Leonard	West Baltimore General Hospital, Baltimore, Maryland
Wanner, Jesse Rosenberger, Jr.	Piedmont Hospital, Atlanta, Georgia
Whitworth, Fuller Barnard	Maryland General Hospital, Baltimore, Maryland
Wilder, Milton Jay	St. Joseph's Hospital, Baltimore, Maryland
Wilner, Sol	Fordham Hospital, New York City
Worsley, Thomas Luther, Jr.	Maryland General Hospital, Baltimore, Maryland
Zalis, Daniel Leonard	Franklin Square Hospital, Baltimore, Maryland

ANNOUNCEMENT OF INTERNSHIP APPOINTMENTS, CLASS OF 1940

Effective July 1, 1940

Algire, Glenn Horner	University Hospital, Baltimore, Maryland
Andrews, Stephen Ralph, Jr.	Church Home & Infirmary, Baltimore, Maryland
Arney, William Charles	Paterson General Hospital, Paterson, New Jersey
Baier, John Cletus	St. Joseph's Hospital, Baltimore, Maryland
Bailey, Walter Levi	York Hospital, York, Pennsylvania
Barker, Daniel Cleveland	Waterbury Hospital, Waterbury, Connecticut
Beacham, Edmund George	Baltimore City Hospitals, Baltimore, Maryland
Biehl, Harold Paul	Maryland General Hospital, Baltimore, Maryland
Borden, Jesse Nachlas	Sinai Hospital, Baltimore, Maryland
Brinsfield, Irving Carlton	Maryland General Hospital, Baltimore, Maryland
Caplan, Lester Harold	University Hospital, Baltimore, Maryland
Chandler, Weldon Porter	North Carolina Baptist Hospital, Winston-Salem, North Carolina
Clifford, Robert Henry, Jr.	Union Memorial Hospital, Baltimore, Maryland
Cole, John Totterdale	Church Home & Infirmary, Baltimore, Maryland
Correll, Paul Harvey	Mercy Hospital, Baltimore, Maryland
Daniel, Louie Samuel	City Hospital, Winston-Salem, North Carolina
Daue, Edwin Oliver, Jr.	University Hospital, Baltimore, Maryland
De Luca, Joseph	St. Mary's Hospital, Waterbury, Connecticut
Dent, Charles Frederic	Western Pennsylvania Hospital, Pittsburgh, Pennsylvania
Don Diego, Leonard Vincent	Coney Island Hospital, Brooklyn, New York
Duffy, William Carroll	Mercy Hospital, Baltimore, Maryland
Dwyer, James Richard	St. Agnes Hospital, Baltimore, Maryland
Freeman, James Albert, Jr.	Mercy Hospital, Baltimore, Maryland
Fusting, William Hammond	Bon Secours Hospital, Baltimore, Maryland
Gassaway, William Farrow	Baltimore City Hospitals, Baltimore, Maryland
Gibbs, Robert Louis	University Hospital, Baltimore, Maryland
Glick, Irving Van der Veere	Harlem Hospital, New York City
Graham, Walter Raleigh	University Hospital, Baltimore, Maryland
Guzman-Lopez, Luis Roberto	University Hospital of the School of Tropical Medicine of the Columbia University, San Juan, Puerto Rico
Hecht, Morton, Jr.	Sinai Hospital, Baltimore, Maryland
Henning, Emil Heller, Jr.	Maryland General Hospital, Baltimore, Maryland
Heyman, Albert	Baltimore City Hospitals, Baltimore, Maryland
Hooton, Elizabeth Louise	Wilkes-Barre General Hospital, Wilkes-Barre, Pennsylvania
Hope, Daniel, Jr.	St. Agnes Hospital, Baltimore, Maryland
Igartua-Cardona, Susana	Bayamon District Charity Hospital, Bayamon, Puerto Rico
Inloes, Benjamin Harrison, Jr.	University Hospital, Baltimore, Maryland
Jamison, William Parks	Western Pennsylvania Hospital, Pittsburgh, Pennsylvania
Jorgensen, Louis Cecil	Dee Hospital, Ogden, Utah
Karns, James Roscoe	University Hospital, Baltimore, Maryland
Kirchick, Julian Gilbert	St. Vincent's Hospital, Bridgeport, Connecticut
Kohn, Schuyler George	Metropolitan Hospital, Welfare Island, New York
Krieg, Edward Louis Joseph	Bon Secours Hospital, Baltimore, Maryland
Kurland, Albert Alexander	Sinai Hospital, Baltimore, Maryland

Lartz, Robert Eshelman	Pittsburgh Medical Center, Pittsburgh, Pennsylvania
Ling, William Soy Ming	Misericordia Hospital, New York City
Livingood, William Cook	Mercy Hospital, Baltimore, Maryland
Loker, Frank Ford	Mercy Hospital, Baltimore, Maryland
Maccubbin, Harry Pearce	University Hospital, Baltimore, Maryland
Markline, Simeon Van Trump	St. Vincent's Hospital, Jacksonville, Florida
Martin, Clarence Wilbur	St. Joseph's Hospital, Baltimore, Maryland
Maryanov, Alfred Richard	Knickerbocker Hospital, New York City
Mathers, Daniel Hutchinson	St. Vincent's Hospital, Jacksonville, Florida
McCann, Harold Francis	Mercy Hospital, Pittsburgh, Pennsylvania
McClung, James Edward	Ohio Valley General Hospital, Wheeling, West Virginia
McClung, William Dennis	Mercy Hospital, Baltimore, Maryland
McDaniel, George Croxton	California Hospital, Los Angeles, California
McKinnon, William James	Maryland General Hospital, Baltimore, Maryland
Meade, Forest Chauncey	Mercy Hospital, Baltimore, Maryland
Miceli, Joseph	St. John's Hospital, Cleveland, Ohio
Molz, Edward Louis	St. Joseph's Hospital, Baltimore, Maryland
Murphy, Frederick Elbert, Jr.	Piedmont Hospital, Atlanta, Georgia
Muse, William Travers	St. Agnes Hospital, Baltimore, Maryland
Myers, George Roger, Jr.	Medical Corps, U. S. Navy
O'Hara, James Francis, Jr.	Mercy Hospital, Canton, Ohio
Pico-Santiago, Guillermo	Presbyterian Hospital, Santurce, Puerto Rico
Pierpont, Ross Zimmerman	Maryland General Hospital, Baltimore, Maryland
Pigford, Robert Toms	Union Memorial Hospital, Baltimore, Maryland
Platt, William	St. Joseph's Hospital, Lexington, Kentucky
Pollock, Arthur Edgar	Mercy Hospital, Altoona, Pennsylvania
Posner, Leonard	Coney Island Hospital, Brooklyn, New York
Pound, John Costello	St. Agnes Hospital, Baltimore, Maryland
Rhode, Charles Martin	University Hospital, Baltimore, Maryland
Richter, Conrad Louis	St. Joseph's Hospital, Baltimore, Maryland
Robinson, Raymond Clarence Vail	Maryland General Hospital, Baltimore, Maryland
Roop, Donald James	Mercy Hospital, Baltimore, Maryland
Rothschild, Carl Eliot	Mount Sinai Hospital, Cleveland, Ohio
Russell, Thomas Edgie, Jr.	Bon Secours Hospital, Baltimore, Maryland
Russillo, Philip Joseph	Misericordia Hospital, New York City
Schlesinger, George Gerard	Morrisania City Hospital, New York City
Sloan, Joseph Wright	Kings County Hospital, Brooklyn, New York
Smith, James Brady	St. Agnes Hospital, Baltimore, Maryland
Smith, Ruby Arden	Charleston General Hospital, Charleston, West Virginia
Squillante, Orlando John	Danbury Hospital, Danbury, Connecticut
Stayton, Howard Nehemiah, Jr.	Delaware Hospital, Wilmington, Delaware
Supik, William Joseph	St. Joseph's Hospital, Baltimore, Maryland
Tankin, Louis Haberer	Sinai Hospital, Baltimore, Maryland
Thompson, Alexander Frank	University Hospital, Baltimore, Maryland
Tompakov, Samuel Victor	Sinai Hospital, Baltimore, Maryland
Townshend, Wilfred Henry, Jr.	University Hospital, Baltimore, Maryland
Trevor, William	Bryn Mawr Hospital, Philadelphia, Pennsylvania
Triplett, William Carryl	Ohio Valley General Hospital, Wheeling, West Virginia
Waite, Merton Theodore	Sibley Memorial Hospital, Washington, District of Columbia
Weeks, William Earl	Mercy Hospital, Baltimore, Maryland

Wilkins, Jesse Lee	Doctors Hospital, Washington, District of Columbia
Williams, Herman Joseph	U. S. Marine Hospital, Baltimore, Maryland
Williams, Richard Talbot	University Hospital, Baltimore, Maryland
Wilson, Harry Thomas, Jr.	St. Joseph's Hospital, Baltimore, Maryland
Wolff, William Irwin	Bellevue Hospital (Second Cornell Medical Division), New York City
Wright, James Rhodes	Maryland General Hospital, Baltimore, Maryland
Zinkin, Solomon Bernard	Morrisania City Hospital, New York City

MATRICULATES

FOURTH YEAR CLASS 1939–1940

Algire, Glenn Horner, B.S.	Maryland
Andrews, S. Ralph, Jr., A.B.	Maryland
Arney, William Charles, B.S.	North Carolina
Baier, John Cletus	Maryland
Bailey, Walter Levi, A.B.	Pennsylvania
Barker, Daniel Cleveland, A.B.	Connecticut
Beacham, Edmund George, B.S.	Maryland
Biehl, Harold Paul, A.B.	Maryland
Borden, Jesse Nachlas, A.B.	Maryland
Brinsfield, Irving Carlton, A.B.	Maryland
Caplan, Lester Harold, A.B.	Maryland
Chandler, Weldon Porter, B.S.	North Carolina
Clifford, Robert Henry, Jr., B.S.	New Jersey
Cole, John Totterdale	Ohio
Correll, Paul Harvey, A.B.	Maryland
Daniel, Louie Samuel, B.S.	North Carolina
Daue, Edwin Oliver, Jr., B.S.	Maryland
De Luca, Joseph, B.S.	Rhode Island
Dent, Charles Frederic	West Virginia
Don Diego, Leonard Vincent, B.S.	New York
Duffy, William Carroll, A.B.	Maryland
Dwyer, James Richard, B.S.	Pennsylvania
Freeman, James Albert, Jr.	West Virginia
Fusting, William Hammond, B.S.	Maryland
Gassaway, William Farrow	Maryland
Gibbs, Robert Louis, A.B.	North Carolina
Glick, Irving Van der Veere, A.B.	New York
Graham, Walter Raleigh, A.B.	North Carolina
Guzman-Lopez, Luis Roberto	Puerto Rico
Hecht, Morton, Jr., B.S.	Maryland
Henning, Emil Heller, Jr., A.B.	Maryland
Heyman, Albert	Maryland
Hooton, Elizabeth Louise, B.S.	Maryland
Hope, Daniel, Jr.	Maryland
Igartua-Cardona, Susana	Puerto Rico
Inloes, Benjamin Harrison, Jr.	Maryland
Jamison, William Parks, B.S.	West Virginia
Jorgensen, Louis Cecil, A.B.	Utah
Karns, James Roscoe	Maryland
Kirchick, Julian Gilbert, A.B.	New York
Kohn, Schuyler George, B.S.	Maryland
Krieg, Edward Louis Joseph	Maryland
Kurland, Albert Alexander	Maryland
Lartz, Robert Eshelman, B.S.	Pennsylvania
Ling, William Soy Ming, A.B.	New York
Livingood, William Cook, B.S.	Pennsylvania
Loker, Frank Ford, B.S.	Maryland
Maccubbin, Harry Pearce, B.S.	Maryland
Markline, Simeon Van Trump, A.B.	Maryland
Martin, Clarence Wilbur	Maryland
Maryanov, Alfred Richard	New York
Mathers, Daniel Hutchinson, A.B.	Maryland
McCann, Harold Francis, A.B.	West Virginia
McClung, James Edward, A.B.	West Virginia
McClung, William Dennis	West Virginia
McDaniel, George Croxton, A.B.	Maryland
McKinnon, William James, A.B.	North Carolina
Meade, Forest Chauncey	Maryland
Miceli, Joseph, A.B.	Maryland
Molz, Edward Louis, B.S.	Maryland
Murphy, Frederick Elbert, Jr., A.B.	Georgia
Muse, William Travers, B.S.	Maryland
Myers, George Roger, Jr., A.B.	Maryland
O'Hara, James Francis, B.S.	Ohio
Pico-Santiago, Guillermo	Puerto Rico
Pierpont, Ross Zimmerman	Maryland
Pigford, Robert Toms	North Carolina
Platt, William	Maryland
Pollock, Arthur Edgar, B.S.	Pennsylvania
Posner, Leonard, B.S.	New York

Pound, John Costello, A.B.	Maryland
Rhode, Charles Martin, A.B.	Maryland
Richter, Conrad Louis	Maryland
Robinson, Raymond Clarence Vail	Maryland
Roop, Donald James, A.B.	Maryland
Rothschild, Carl Eliot, B.S.	China
Russell, Thomas Edgie, Jr., B.S.	Maryland
Russillo, Philip Joseph, A.B.	New Jersey
Schlesinger, George Gerard, B.S.	New York
Sloan, Joseph Wright, B.S.	New Jersey
Smith, James Brady, B.S.	Maryland
Smith, Ruby Arden	West Virginia
Squillante, Orlando John, B.S.	Rhode Island
Stayton, Howard Nehemiah, Jr., A.B.	Delaware
Supik, William Joseph	Maryland
Tankin, Louis Haberer, A.B.	Maryland
Thompson, Alexander Frank, A.B.	North Carolina
Tompakov, Samuel, A.B.	Maryland
Townshend, Wilfred Henry, Jr., A.B.	Maryland
Trevor, William, A.B.	Maryland
Triplett, William Carryl	West Virginia
Waite, Merton Theodore	Maryland
Weeks, William Earl, B.S.	North Carolina
Wilkins, Jesse Lee, B.S.	Maryland
Williams, Herman Joseph, B.S.	Pennsylvania
Williams, Richard Talbot, A.B.	Maryland
Wilson, Harry Thomas, Jr.	Maryland
Wolff, William Irwin, B.S.	New York
Wright, James Rhodes, A.B.	North Carolina
Zinkin, Solomon Bernard, A.B.	New Jersey

THIRD YEAR CLASS 1939–1940

Alberti, Aurora Frances, A.B.	New York
Alexander, Fred, A.B.	New Jersey
Arnett, Jerome Cayton, B.S.	West Virginia
Barnett, Charles Phelps, A.B.	Maryland
Baxley, Joshua Warfield, III, B.S.	Maryland
Bowen, Joseph John, B.S.	Connecticut
Brooks, Julius Culpepper, Jr., B.S.	Tennessee
Bundick, William Ross	Maryland
Carter, John Marshall, A.B., B.S.	West Virginia
Checket, Pierson Melvin, A.B.	Maryland
Chiqués, Carlos Miguel	Puerto Rico
Cloninger, Charles Edgar, A.B.	North Carolina
Conlen, Richard Alexis, B.S.	New Jersey
Cooper, LeRoy Gerald, A.B.	Pennsylvania
Crecca, Joseph Vincent, B.S.	New Jersey
Croce, Gene Albert, B.S.	Rhode Island
Cruikshank, Dwight Phelph, Jr., A.B.	West Virginia
Culler, John McCleary, A.B.	Maryland
deVincentis, Michael Louis, B.S.	Maryland
Diez-Gutierrez, Emilio	Puerto Rico
DiPaula, Anthony Francis, A.B.	Maryland
Esnard, John Edward	California
Evola, Camille Mary, A.B.	New York
Frey, Edward Leonard, Jr., A.B.	Maryland
Garcia-Blanco, José	Puerto Rico
Gelber, Julius, A.B.	New York
Goodman, William, A.B.	Maryland
Graziano, Theodore Joseph, A.B.	Maryland
Hedrick, Thomas Ardis	West Virginia
Hershner, Newton Webster, Jr., B.S.	Pennsylvania
Hollander, Asher, A.B.	Maryland
Huffman, Pearl Trogdon, A.B.	North Carolina
Hunter, James Stanley, Jr.	Maryland
Jaffe, Vita Rebecca, B.S.	New York
Kemp, Norval Foard, A.B.	Maryland
Krulevitz, Keaciel Kenneth, B.S.	Maryland
Lach, Frank Edward, B.S.	New Jersey
Leslie, Franklin Earl, A.B.	Maryland
Levinson, Lorman Leon, A.B.	Maryland
Licha, José Salomon	Puerto Rico
Lowe, William Cook, B.S.	Maryland
Lusby, Thomas Frank, A.B.	Maryland
Malouf, Raymond N., B.S.	Utah
Mandel, Jacob Barry, M.S.	New Jersey
Mitchell, William Arthur	Maryland
Molinari, José Gilberto	Puerto Rico
Morgan, Margaret	Indiana
Morris, Felix Raymond, B.S.	Connecticut
Morrison, William Herbert	Maryland
Nolan, James Joseph, B.S.	Maryland
Novoa-Caballero, Miguel	Puerto Rico
Ortiz, Idalia Ortiz, B.S.	Puerto Rico
Palmer, Margaret Virginia, M.A.	Maryland
Pasamanick, Benjamin, A.B.	New York
Pearcy, Thompson, A.B.	West Virginia
Perman, Joshua Melvin, A.B.	Maryland
Phrydas, Irene, A.B.	North Carolina
Pruitt, Charles Eugene, A.B.	Maryland
Renna, Francis Stanley, A.B.	New Jersey
Revell, Walter Jones, B.S.	Georgia
Richardson, Charles, Jr.	Maryland
Richmond, Marion Ballard, B.S.	Maryland

Richter, Christian Frederick, Jr., B.S. Maryland
Rosenberg, Jonas Samuel, A.B. New York
Rossberg, Clyde Arthur, A.B.... Maryland
Sasscer, Robert Bowie, A.B..... Maryland
Sawyer, William Hamilton, A.B. North Carolina
Scholl, John Andrew, A.B., B.S. West Virginia
Schwartz, Stanley Eugene, B.S. New York
Seigman, Edwin Lincoln, Jr., A.B. Maryland
Shannon, Edward Patrick, Jr., B.S. New York
Sheehan, Joseph Chester, B.S.... Maryland
Sherrill, Elizabeth Brown, B.S... Maryland
Sims, Thomas Courtland, A.B., B.S. West Virginia
Skitarelic, Benedict, A.B., B.S. West Virginia
Spencer, Tracy Neil, Jr., A.B. North Carolina
Spinnler, Henry Robert, B.S... New Jersey
Stevens, John Sutehall, A.B... Connecticut
Strayer, Webster Mills, Jr., A.B. Maryland
Thompson, Raymond Kief, B.S.. Maryland
Trevaskis, Richard White, A.B. Maryland
Ulrich, George John, A.B........ Maryland
Virusky, Edmund Joseph, B.S. Pennsylvania
Walker, James Haward, A.B. West Virginia
Wall, Lester Aubrey, Jr., A.B... Maryland
Watkins, Dayton O'Lander, B.S. Maryland
Wells, John Bernard, Jr., B.S.... Maryland
Wilder, Thomas Carroll....... Minnesota
Wilson, Edwin Forrest, A.B..... New York
Yanagisawa, Kazuo, A.B....... California
Young, John David, Jr......... Maryland
Zierler, Kenneth Levie, A.B..... Maryland

SECOND YEAR CLASS 1939-1940

*Adam, Alberto Lotfalla...... Puerto Rico
Ahroon, William Alstrom, A.B... Maryland
Bacharach, David Nathan, Jr., A.B. Maryland
Barthel, Robert Amthor, Jr...... Maryland
Bassan, Morton Edward, A.B.... Maryland
Bennett, Van Boring, A.B. North Carolina
Bird, Joseph Gordon, B.S....... Maryland
Bowen, Francis Dorsey Thomas, B.S. Maryland
Brodsky, Alexander Emmanuil, B.S. Maryland
Byerly, William Luther, Jr., B.S. South Carolina
Carey, Richard Alexius, B.S..... Maryland
Coffman, Harry Franklin, II, A.B. West Virginia
Concilus, Frank, A.B........ Pennsylvania
Cox, Matthew Mordecai........ Maryland
Crane, Warren Eugene, B.S.... New Jersey
Davies, Thomas Eugene..... Pennsylvania
Davila-Lopez, José G., B.S.... Puerto Rico
Davis, John Russell, Jr., A.B. West Virginia
Day, Newland Edward......... Maryland
Dillinger, Karl Anthony.... West Virginia
Fallin, Herbert Kirk, A.B....... Maryland
File, Richard Cushing, B.S......... Illinois
Franz, John Howard, A.B...... Maryland
Friedman, Marion, B.S......... Maryland
Fuertes, José Rodriguez, B.S. Puerto Rico
Furnari, Joseph Charles, B.S. Pennsylvania
Goldsmith, Jewett, A.B........ Maryland
Gramse, Arthur Edward, A.B. Massachusetts
Greer, Margaret Alta, B.S...... Maryland
Gregory, Exie Mildred..... West Virginia
Hamburger, Morton Leonard, B.S. Maryland
Hubbard, Prevost, Jr., B.S..... New York
Ingram, Albert Lester, Jr., B.S. Delaware
Irwin, Robert Clark, B.S...... New Jersey
Jones, Everett Davis, A.B...... Maryland
Kardash, Theodore, B.S........ Maryland
Keeley, Joseph Francis, Jr., A.B. Connecticut
Kenyon, Harold Arthur, A.B. Massachusetts
Kiefer, Robert Allan, A.B....... Maryland
Klijanowicz, Stanley Benedict, Ph.B. Maryland
Koleshko, Lawrence Jacob, B.S. Connecticut
Krepp, Martin William, Jr...... Maryland
Kroll, John Gregory, A.B.... Pennsylvania
Kundahl, Paul Charles......... Maryland
Link, Etta Carolyn, B.S......... Maryland
Longwell, Robert Hamilton, B.S. Pennsylvania
Lowitz, Irving Robert, B.S...... Maryland
Lyons, Mary Louise, A.B........... Ohio
Manganiello, Louis Otto, A.B. Connecticut
Marino, Frank Sebastian, A.B. Connecticut

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* Did not complete the year.

Matthews, Henry Steadman, A.B. North Carolina
Mazer, Robert, B.S............Maryland
McCosh, James Nathaniel, Jr., A.B. Maryland
McGoogan, Malcolm Thomas, Jr...Georgia
Meli, John James...........Pennsylvania
Miller, Edgar Allen, Jr., A.B. Pennsylvania
Moses, Robert Abram, A.B.....Maryland
Mullins, George Roy, Jr.....West Virginia
Orofino, Caesar Francis, B.S....New York
Osborne, John Carlton, B.S.....Maryland
Phelan, Patrick Carey, Jr., A.B. Maryland
Phillips, Otto Charles, A.B......Maryland
Posey, Dale Morton, B.S....Pennsylvania
Ritchings, Edward Peyton, A.B. Maryland
Rosin, John David, A.B........Maryland
Rousos, Anthony Peter, A.B....New York
Sadler, Henry Harrison, Jr., A.B. Maryland
Sadowsky, Wallace Hyman, B.S..Maryland
Sborofsky, Isadore, B.S.........Maryland
Scott, Joseph Whiddon, A.B.......Florida
Shipley, Edgar Roderick, A.B...Maryland
Shub, Maurice Isaac, A.B.......Maryland
Shuman, Louis Harry, A.B......Maryland
Stegmaier, James George, B.S...Maryland
Summa, Andrew Anthony James, A.B. New York
Townsend, Francis James, A.B. Maryland
Traynor, Francis Willoughby, B.S. Maryland
*Van Lill, Stephen Joseph, III, A.B. Maryland
Wallace, Joseph, Jr., A.B....Pennsylvania
Ward, Charles Monroe, B.S..West Virginia
Williams, Charles Herman, A.B. Maryland
Williamson, Edgar Percival, Jr., A.B. Maryland
Zimmerman, Loy Miller, B.S....Maryland

FIRST YEAR CLASS 1939–1940

Allsopp, Richard Charles....Pennsylvania
Almodovar, Ramon Ignacio..Puerto Rico
Baker, Emory Forester, A.B..Washington
Barnes, John David, A.B...Massachusetts
Beall, William Lee, A.B.....West Virginia
Bowen, Charles Vernon, Jr., B.S. Maryland
Brennan, Thomas Joseph, B.S..Maryland
Brooks, Ralph King, A.B...West Virginia
Brooks, Ross Chilton, B.S.....Maryland
Bryson, William James, A.B....Maryland
Chenowith, Ralph Stallings....Maryland
Cohen, Harry, B.S............Maryland
Coughlin, John Benedict, B.S. Pennsylvania
Courtney, Donald Lawrence......Oregon
Crastnopol, Philip, B.S......New Jersey
Cusani, Benedict Albert, B.S....Florida
Dalmau, Miguel Sebastian, B.S. Puerto Rico
Davis, William Joseph Graham, A.B. District of Columbia
Deegan, Robert Thomas, A.B.......Ohio
De Rosa, Hugo Anthony, A.B..New Jersey
Diorio, John Daniel, B.S....Connecticut
Dippel, Francis Xavier, B.S....Maryland
Dunne, Thomas Benjamin, A.B.New York
Epperson, John Wallace Walker Maryland
Fowler, Richard Lowman, B.S. North Carolina
French, Samuel Lawson, A.B...Maryland
Friedman, Paul Norman, A.B..Maryland
Garrison, Alfred Selman, A.B..Maryland
Giglia, Tony Robert, Jr., A.B. West Virginia
Grave de Peralta, Jose Ignacio, B.S..Cuba
Gray, David Benoni, B.S...West Virginia
Hagan, William Baker........Maryland
Hassler, Frank Stanley, III, A.B. Pennsylvania
Haydu, Joseph Frank......West Virginia
Honigman, Alvin Herbert, B.S..Maryland
Hutson, Paul Gilliss..........Maryland
Koleshko, William Nickifor, B.S. New Jersey
Lacher, George Milton, B.S....Maryland
La Mar, Robert Charles, Jr....Maryland
Lewis, Richard Quarles, A.B...Maryland
Livingstone, Robert Charles, A.B. West Virginia
Lukats, Paul George..............Ohio
MacDonald, Charles Renwick..Maryland
*Mace, Carville Veirs, Jr., A.B..Maryland
Matchar, Joseph Charles, A.B..Maryland
McMillan, Marcy Emory, Jr. West Virginia
Mele, Vincent James, Jr., B.S..New Jersey
Mendez, Nestor Hernan, B.S..Puerto Rico
Miller, James Delmar, A.B........Ohio
Minervini, Robert Virginius, A.B. New York
Musnick, Henry, A.B.........Maryland
Myers, Joseph Carl, A.B......Maryland
Nash, Kenneth Powell........California
Neff, Charles Amos, A.B........Illinois
Parés, Maria Amalia, B.S.....Puerto Rico
Perez, Enrique, A.B..........Puerto Rico
Peterson, Preston Horsley..........Utah

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* Did not complete the year.

Queen, Joseph Emmett, A.B....Maryland
Rangle, Raymond Veto........Maryland
Renshaw, Josephine Elizabeth, M.S. Maryland
Richards, Granville Hampton, A.B. Maryland
Robbins, Martin Albert, A.B...Maryland
Rosenstein, Louis Nathan, B.S..Maryland
Royer, Earl Linwood..........Maryland
Rude, Richard Sprogoe......New Jersey
Sacks, Seymour, A.B..........Maryland
Samuels, Irving Leonard, A.B...Maryland
Sharp, Nathaniel.............Maryland
Sigler, John Wiltshire, A.B..........Ohio
Soler-Favale, Marta Emilia, B.S. Puerto Rico
Spier, Andrew Allan, A.B......Maryland
Stafford, Harold Rellinger.....Michigan
Stewart, Edwin Harvey, Jr....Maryland
Stier, Howard William, A.B..New Jersey
Stoner, James Ernest, Jr., A.B..Maryland
Taylor, Irving Julian, A.B.....Maryland
Tilt, LeRoy Wortendyke, Jr., A.B. Maryland
Torres, José Manuel, B.S....Puerto Rico
Trader, Charles Weldon, A.B...Maryland
Trent, Letcher Evans, Jr., A.B..Kentucky
Tunney, Robert Boone, B.S....Maryland
Varhol, Joseph Gregory, Jr., B.S. New Jersey
Wachsman, Irvin Louis, B.S....Maryland
Walker, Samuel Haywood, B.S. North Carolina
Warren, Frank Orville, Jr..New Hampshire
Webster, Thomas Clyde, B.S....Maryland
Weiss, Maurice Richard....Pennsylvania
Wich, Joseph Carlton, B.S.....Maryland
Wilson, Thomas Leslie........Maryland
Wise, Robert Edward....Pennsylvania
Wishbow, Eleanor Marie, A.B. New Jersey
Wooddy, Arthur Overton......Maryland
Worgan, David Kuykendall....Maryland
Wysong, William Scott, Jr..West Virginia
Yurko, Leonard Emory, A.B.West Virginia

LIST OF COLLEGE SOURCES OF STUDENTS ENROLLED DURING 1939–1940*

Akron University	1
Alabama Polytechnic Institute	2
Albright College	1
Alliance Junior College	1
American University	2
Antioch College	2
Baylor University	1
Beckley Junior College	1
Bethany College	1
Blue Ridge College	3
Bridgewater College	2
Brooklyn College	1
Bucknell University	1
California, University of	1
Campbell College	1
Catholic University of America	1
Chattanooga, University of	1
Chicago, University of	2
Citadel, The	1
Columbia Junior College	2
Columbia University	6
Concord State Teachers College	1
Cornell University	2
Dartmouth College	1
Davidson College	3
Delaware, University of	1
Dickinson College	2
Duke University	2
Emory Junior College	1
Emory University	1
Erskine College	1
Fairmont State Teachers College	1
Florida Southern College	1
Fordham University	1
Franklin and Marshall College	1
Georgetown University	3
George Washington University	1
Georgia, University of	2
Gettysburg College	2
Goshen College	2
Goucher College	1
Harvard College	5
Holy Cross College	1
Hunter College	1
Illinois, University of	1
Indiana University	2
Iowa State University	1
Johns Hopkins University	57
Juniata College	2
Kentucky, University of	1
La Salle University	1
Lenoir-Rhyne College	2
Louisville, University of	1
Loyola College	19
Loyola University	1
Manhattan College	1
Marshall College	3
Mars Hill College	2
Maryland State Normal School	1
Maryland State Teachers College	1
Maryland, University of	86
Massachusetts Institute of Technology	1
McGill University	1
Meredith College	1

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* Totals exceed student total because of multiple college attendance.

Michigan, University of	1
Middlesex Junior College	1
Minnesota, University of	1
Mount St. Mary's College	3
Muhlenberg College	1
Muskingum College	1
New Hampshire, University of	1
New River State College	1
New York, College of the City of	3
New York University	14
Niagara University	1
North Carolina, University of	13
Northwestern University	1
Notre Dame, University of	2
Oak Ridge Military Institute	1
Oberlin College	1
Ohio State University	2
Oregon, University of	1
Pennsylvania State College	2
Pennsylvania State Teachers College	1
Pennsylvania, University of	1
Pittsburgh, University of	5
Portland, University of	1
Potomac State College	1
Princeton University	1
Puerto Rico, Polytechnic Institute of	1
Puerto Rico, University of	16
Randolph-Macon College	1
Rhode Island State College	3
Roanoke College	1
Rochester, University of	1
Rutgers University	1
St. Elizabeth, College of	1
St. Francis College	1
St. John's College	16
Seton Hall College	1
Smith College	1
South Carolina, University of	1
Southern California, University of	1
Springfield College	3
Syracuse University	1
Tennessee, University of	1
Transylvania College	1
Tri-State College	1
Tufts College	1
United States Military Academy	1
Utah State Agricultural College	1
Utah, University of	2
Villanova College	6
Virginia Polytechnic Institute	1
Virginia, University of	1
Wake Forest College	4
Washington College	5
Washington and Jefferson College	1
Washington and Lee University	4
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Wayne University	1
Weaver College	1
Wellesley College	1
Wesleyan College	1
Western Maryland College	15
West Virginia State Teachers College	1
West Virginia University	26
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William and Mary, College of	1
Wilson College	1
Wisconsin, University of	1
Yale University	1
Yenching University	1

GEOGRAPHICAL DISTRIBUTION OF STUDENTS, 1939–1940

California	3
Connecticut	10
Delaware	2
District of Columbia	1
Florida	2
Georgia	3
Illinois	2
Indiana	1
Kentucky	1
Maryland (Baltimore 101)	182
Massachusetts	3
Michigan	1
Minnesota	1
New Hampshire	1
New Jersey	20
New York	23
North Carolina	19
Ohio	7
Oregon	1
Pennsylvania	24
Rhode Island	3
South Carolina	1
Tennessee	1
Utah	3
Washington	1
West Virginia	31

FOREIGN

China	1
Cuba	1
Puerto Rico	21

SUMMARY OF STUDENTS—1939-1940

Class	*Men*	*Women*	*Total*
Senior	97	3	100
Junior	84	8	92
Sophomore	79*	4	83*
Freshmen	91†	4	95†
	351	19	370
Withdrawals, *2, †1	3	0	3
	348	19	367

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They may be made to the general or University Fund, to the Medical Fund or to any other department of the University. If intended for the School of Medicine, they may be given to the general medical fund or to some special object, as building, research, library, pathology, hospital, publication, laboratories, gymnasium, scholarship, medal, prize, etc., in which case the wishes of the donor will be strictly regarded. Attention is invited to the "Charles Frick Research Fund," already established in memory of that distinguished investigator. Checks should be made payable to Endowment Fund of the University of Maryland, J. M. H. Rowland, Treasurer, Lombard and Greene Streets, Baltimore, Md.

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I give, devise and bequeath to the Regents of the University of Maryland, a corporation incorporated under the laws of the State of Maryland, for the benefit of the Faculty of Physic..

(Here state amount or describe property)

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(Here state amount or describe property)

THE UNIVERSITY OF MARYLAND SCHOOL OF NURSING

The University of Maryland School for Nurses was established in the year 1889. Since that time it has been an integral part of the University of Maryland, coming under the same government. It is a non-sectarian school, the only religious services being morning prayers.

The University Hospital is a general hospital containing about 435 beds. It is equipped to give young women a thorough course of instruction and practice in all phases of nursing.

Programs Offered: The programs of study of the school are planned for two groups of students: (a) the five-year group, (b) the three-year group.

The requirements for admission to the five-year program of the School of Nursing are the same as for other colleges of the University. The completion of this course entitles the student to the degree of Bachelor of Science from the University of Maryland and to the diploma of the University Hospital School of Nursing.

The requirements for admission to the three year program are the satisfactory completion of an academic course in an approved high school. The completion of this course entitles the student to the diploma of the University of Maryland School of Nursing. Blank certificates will be furnished upon application to the Director of the School of Nursing.

MERCY HOSPITAL SCHOOL OF NURSING

The Mercy Hospital School of Nursing was established in 1899 and incorporated on December 23, 1901. It has developed the art of the profession according to the high standard necessary to qualify as a Registered Nurse.

The rapid growth of the institution, attested by the increasing number of its graduates, is evidence of the active part it takes in the health of the community. Through its connection with the Mercy Hospital its opportunities are unlimited. By its affiliation with the University of Maryland it has the advantage of the best professors for the instruction of the nurses.

The University of Maryland, in affiliation with the Mercy Hospital School of Nursing, offers a five year combined Academic and Nursing program. The completion of this course entitles the student to the degree of Bachelor of Science from the University of Maryland, and to the diploma of the Mercy Hospital School of Nursing. Graduate nurses who hold college degrees are greatly in demand, especially for positions in administration and teaching institutions. This program consequently offers a distinct advantage.

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